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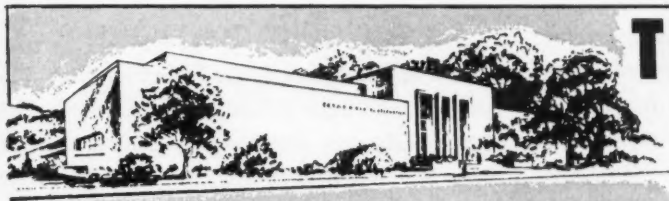

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LIVING PRINCIPLES

Resolutions and a new year are as inseparable in the American mind as bread and butter. With the twelve months now beginning, perhaps no better resolution could be adopted by each American than the determination to *apply* the principles of government, economics, and social behavior to which most Americans automatically subscribe.

The men who framed the Constitution of the United States provided for a representative democracy based on broad participation by the citizenry. They set up checks and balances between the executive, legislative, and judicial arms of the federal government to keep one branch from usurping power. They provided for a system of free economic enterprise in which private property could be amassed by individuals from whom it could not be taken without due process of law; for free export and import of articles from state to state; and for a three-way system of economic control in which the free employer, the free worker, and the free customer counterbalance each other. Finally, a bill of rights to guarantee such freedoms as worship, speech, assembly, and press reports was made a part of the Constitution. Between its numbered paragraphs, although not in so many words, were the undergirding moral and ethical principles of the great religions, without which such a constitution could but fail.

Those who labored in Constitution Hall to draw up a document to shape the future of the new republic had doubts as to whether the people actually were capable of self-government. Benjamin Franklin said: "You now have a republic—if you can keep it." He and the others who earlier had penned the Declaration of Independence, in stating one inalienable right to be "the pursuit of happiness," recognized that happiness cannot be conferred, that happiness and security must be created by each who seeks it. This is still true.

Blessed as we are by being part of a land where great principles have been laid down, our greatest liability is lethargy and the perhaps un-

Poll Taxes Are Payable Before January 31

conscious reluctance to accept these principles for application in our own individual situations.

We cannot have a government by the people unless we in Texas take the trouble to pay our poll taxes and then vote on election day. We cannot have individual freedom while condoning irresponsible name-calling or intolerance. We cannot apply one rule to "the other fellow" and another to ourselves.

The beginning of a new year should be a time of soul searching. If we are in earnest about believing in government of the people, by the people, and for the people; if we honestly think that free enterprise can bring greater goods and services to more people; if deep down in our hearts we have concern for all our fellow creatures—if we pay homage to such principles, let us resolve to live them in this year ahead.

JOURNAL ARTICLES INVITED

For years the bulk of the scientific articles published in the TEXAS STATE JOURNAL OF MEDICINE have been presented first on the program of the annual session of the Texas Medical Association. The typical annual session in recent years has yielded up to 100 essays, of which the majority have been published in subsequent issues of the JOURNAL. The 1954 annual session is being planned around a new format which will offer perhaps half as many papers for possible publication.

Mathematicians will conclude correctly that during the forthcoming year a greater opportunity than ever for acceptance of direct contributions to the JOURNAL will prevail. Nevertheless, it is not expected that the procedure of screening manuscripts or the high standards for acceptance will be modified.

The editorial staff and its advisers will be pleased in the next few months to review papers which were well received at a district or county medical society meeting or which were prepared especially for the JOURNAL. Suggestions for preparation of manuscripts may be obtained from the JOURNAL office upon request.

THE FIGHT CONTINUES

In 1953, Texans contributed more than $2,500,000 to fight poliomyelitis through the March of Dimes. The National Foundation for Infantile Paralysis is now staging its 1954 drive, January 2-31, to conquer poliomyelitis by raising money for personnel, equipment, facilities, and medical research.

Progress toward the conquest of the dreaded disease is being made, but at a terrific cost. Gamma globulin has been found to provide marked temporary protection against paralytic poliomyelitis and possibly to modify the severity of the disease during the late stages of the incubation period, but gamma globulin research must continue. More recently, a vaccine has been developed. Its effectiveness in preventing paralytic poliomyelitis will be tested in a mass vaccination project beginning in February. The estimated cost of $7,500,000 for this study will be financed by the national foundation.

Since 1938, when the national foundation was brought into being, 87 per cent of all March of Dimes collections in Texas has been used to care for patients in the state and only 13 per cent to assist in the national program.

To date, the National Foundation for Infantile Paralysis has given more than $200,000 in research grants to six Texas institutions, and the Southwestern Poliomyelitis Respiratory Center at Houston is one of six in the United States supported by March of Dimes funds. In addition, more than 3,000 scholarships and fellowships, forty-five to Texas residents, have been given by the national foundation.

Physicians have a wide scope of friends and acquaintances whom they can urge to support the drive as they themselves are doing. They can encourage their wives to join the Mothers' March on Polio, and their children can be useful in school drives. In addition, they can make every effort to learn more about the disease, its cause and cure, and do their share to halt poliomyelitis in the near future.

CURRENT EDITORIAL COMMENT

THE PHYSICIAN, CRIME, AND THE COURT

The medical profession has never been interested in the social and economic problems referable to crime. However, today it seems wise for us, as good citizens of the community and as members of a profession which must treat both the criminal and his victim without being influenced by either guilt or innocence, to acquaint ourselves with these problems.

It has been estimated that the national bill for crime is $28 billion; of this $20 billion is for gambling, $7.5 billion for organized crime including racketeering, and $500 million for conventional crimes. This is a multibillion dollar drain upon our national resources; the slogan "Crime Does Not Pay" apparently is not proving very effective. Statistics show that young people are major contributors to crime. In 1950, 30.5 per cent of arrests were of persons younger than 25 years of age. These youths were charged with 54.1 per cent of robberies, 61.6 per cent of burglaries, 45.4 per cent of larcenies, and 67.3 per cent of automobile thefts. Approximately one-half of the crimes against property were said to have been committed by members of this same age group.

The interest of lawyers in familiarizing themselves with medicolegal problems, as indicated by their participation in the law-science program sponsored by the Schools of Law and Medicine of the University of Texas, should be a challenge to the medical profession. A physician frequently has to appear in court as a witness on behalf of either the prosecution or the defense. Without knowledge of legal procedures he often is embarrassed by questions propounded by the opposing attorneys. He frequently does not take time to become familiar with the legal processes and medical problems involved before going on the witness stand. The results are not only embarrassment to himself but failure to convey to the jury a clear concept of the problems on which it is to render a verdict. Apparently it will be necessary for us, as physicians, to appear in courts more and more, and it behooves all of us to meet this responsibility by increasing our knowledge of legal procedure as it applies to expert testimony. Insufficient knowledge of the case, poor techniques in presenting testimony, and testimony by unscrupulous doctors will cause the public quickly to lose faith in the medical profession.

A few physicians as pathologists and toxicologists contribute to the solution of crimes. The great need today, however, is prevention of crime and institution of research to determine better methods of punishment. The medical profession is in a better position than any other group to contribute constructively to these programs. Such efforts must not be considered only in the fields of the pathologist and the psychiatrist. Malnutrition, disease, and physical defects are medical problems frequently affecting the lives of persons who are either criminals or potential criminals. It has been said that "the most inspiring experiment of modern times in criminology and penology is that proceeding at the new Federal Correctional Institution at Seagoville, Texas." (Seagoville is a "prison without walls" with a minimum of physical and mental barriers. There one finds an atmosphere relaxing, alert, purposeful, and calm instead of the usual one of tenseness, intrigue, repression, and regimentation.) Thus, we see that the seeds of research are growing in our state. As a profession, let us contribute our part in the cultivation of these efforts and actively assist in the development of sociologic programs aimed at the prevention of crime.

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ORIGINAL ARTICLES

THE ANTICOAGULANTS

MARVIN DUNN, RAYMOND HENRY, JOHN RIBBLE, CHARLES SANDERS, and K. P. WALKER, Dallas, Texas

Under the Direction of Andres Goth, M. D., Dallas, Texas

UNTIL the past few years, the fight of the physician against thromboembolic disease has been based on two procedures, venous ligation and early ambulation. These devices have met with only limited success because of the widespread basic dysfunction in the vascular tree. Thromboembolic diseases are based on a number of interrelated factors,[56] among which are (1) generalized lesions of the vascular tree; (2) pressure changes and rate of blood flow; (3) characteristics of the formed elements; and (4) coagulability of the plasma. All these are so balanced as to cause the blood to remain fluid in its channels and also to prevent extravasation of fluid from the capillary network. Any interruption of this in a positive direction likely will result in intravascular thrombosis, while tilting the balance the other way might well prevent such phenomena.

Indeed, there now are available to the medical profession such anticoagulants which may be used in thromboembolic disease. A group of Scandinavian workers in 1935 were the first to present clinical data that such drugs were of definite therapeutic use.[33] Led by Jorpes, these clinicians administered anticoagulants to some 900 patients, 138 of whom they claimed would have died without their use.

There is one factor which must be considered in the administration of anticoagulants. Improperly controlled therapy will lead to hemorrhage. The incidence of such bleeding ranges from 0 to 33 per cent of all cases treated with anticoagulants.[56] However, as research progresses, an understanding of the various drugs has come about with subsequent alterations in the mode of treatment. The main guide in all therapy is based on the calculation of coagulation and prothrombin times. It is well to mention that prothrombin time is used only in patients being treated with drugs affecting liver function—for example, Dicumarol—while clotting time is instrumental in determining the action of such drugs as heparin.

Clotting time is measured by a number of methods including: the Lee-White, the Howell, and the capillary methods. They are all based on the same four factors: (1) the sample should not be contaminated with tissue substance; (2) clean glassware should be used; (3) room temperature should be optimal for determination; and (4) consistent amounts of trauma and surface contact should be exerted on every part of the sample, if any degree of accuracy is to be achieved. Prothrombin is a plasma protein chemically behaving as a euglobulin. Its production is thought to be solely in the liver, with vitamin K playing an important role in its manufacture. Activity of this protein is determined by taking oxalated plasma and adding relatively large amounts of ionized calcium and thromboplastin.[45] In this way all the clotting factors are in excess with the exception of prothrombin, and the clotting time then is a reflection of the prothrombin concentration. This test is often performed on diluted plasma (usually 12.5 per cent), since this adds greater sensitivity to the reaction with accordingly more accurate results.[2]

Proper treatment requires an understanding of the coagulation mechanism. Although there is a great deal of controversy over the various factors involved, the Quick theory, for the sake of expediency, will be discussed herein, since it provides the necessary framework in explaining the physiologic action of these drugs. Quick postulated that some damage to the endothelium causes lysis of the platelets, liberating probably thromboplastinogenase, which reacts with a plasma factor, probably thromboplastinogen, to form thromboplastin. This product added to ionized calcium, accelerator globulin, and prothrombin results in thrombin, an enzyme which allows fibrinogen to form fibrin. Thrombin also causes lysis of platelets and, thus, the cycle repeats itself.

The concept of treating thromboembolic disease by employing methods which afford diminished coagulability represents a physiologic approach to the problem. Indeed, it may even be considered an attack on the etiologic agent, though truly this would involve elimination of the factor responsible for the disease. Nevertheless, this approach seems to be the most desirable, for the more closely a therapeutic effort fits into the body physiology, the greater the chance for its success without toxic effects.

The authors are students at Southwestern Medical School of the University of Texas and prepared the article under the direction of Andres Goth, M. D., professor of pharmacology.

The relationship between coagulability of blood and thrombosis is not clear. Similarly, the mechanism of anticoagulant action is obscure. Anticoagulants seem to act in one of the following ways:[54] (1) decreased metabolic production of a coagulant; (2) increased metabolic production of an anticoagulant; (3) direct inactivation or destruction of a clotting factor; or (4) release of a stored anticoagulant. Though it is thought that Dicumarol (one of the major anticoagulants) acts to decrease metabolic production of a coagulant and that heparin (a second major anticoagulant) produces direct inactivation or destruction of a clotting factor,[4] there is no conclusive proof that these are the only important actions of these drugs. Much still is unexplained, and the avenue of investigation is the only road to such knowledge.

Therapeutic indications for the anticoagulants in general are (1) acute thrombophlebitis; (2) phlebothrombosis; (3) recurrent thrombophlebitis; (4) pulmonary and peripheral thromboembolism; (5) prophylaxis to prevent thromboembolism postpartum, postoperatively (particularly in pelvic surgery), and when threatening thrombosis is suggested by hypercoagulability and previous history of thrombosis; (6) coronary thrombosis and myocardial infarction; and (7) diseases with multiple small vessel thrombosis.[56]

HEPARIN

Heparin was discovered accidentally[34] by Jay McLean of Johns Hopkins University in 1916 while he was working with William Howell. It was first isolated from heart and liver[34] and given the name of heparin in 1918 by Howell and Holt[28] because of its abundant occurrence in the liver. By 1924 Howell had so purified the product that 1 mg. would prevent coagulation of 40 cc. of cat blood at 0 C. for twenty-four hours.

Heparin is a normal physiologic compound found in the metachromatic granules of the mast cells of the body[35] and is particularly abundant in the liver and lungs. These cells are located near blood vessels and capillaries, and the contents of the granules can be emptied directly into the blood stream.[35] The heparin structure is that of a mucoitin polysulfuric acid with a disaccharide repeating unit in the molecule. The molecular weight is in the neighborhood of 17,000.[35] Heparin is one of the body's most acidic substances,[3] but mild activity causes the molecule to lose its anticoagulant activity.[54] It carries amino groups with strong negative charges[54] which are essential to its activity.[53]

Heparin is not absorbed from the gastrointestinal tract and is not effective if administered sublingually.[43] Intravenous, intramuscular, or subcutaneous injections are the only effective means of giving the drug. Repository forms and intravenous drip have been used to circumvent the usual rapid disappearance of the drug from the circulation.

Heparin is probably inactivated by an enzyme,[54] since it is eliminated so rapidly, but it is also excreted in the kidneys to some extent as uroheparin.[7] This may be of only slight importance as it has been shown to disappear just as rapidly in nephrectomized animals.[6] The extent of a heparin-inactivating enzyme is unconfirmed, but heparin-protein inactivating complexes have been described,[4] though also unconfirmed. It has been proposed that body resynthesis of thromboprotein removes heparin, thus eliminating excretion, enzymes, and protein complexes as routes of dissipation.[4] The use of carinamide to reduce tubular excretion by the kidneys has been shown to increase the duration of heparin action.[41]

Deposits of heparin have been found in the liver, lung, spleen, and kidneys of experimental animals.[54] However the main supply remains in the blood.

The exact mechanism of action of heparin has not been established fully, but it is known that it acts before fibrinogen can proceed to fibrin in the clotting process. It has been shown to act at three points: (1) blocking the activation of prothrombin, probably through a plasma cofactor;[51] (2) acting as an antithrombin, also most likely through a plasma cofactor;[26] and (3) producing a decrease in platelet number[54] and preventing their agglutination,[51] possibly through the action of the first two mechanisms.[54] These effects on the blood clotting process produce prolonged clotting times.[32] One theory for the action of heparin is that by combination in the tissues with a thromboprotein and lipid substance, the heparin releases another factor which has been shown to be a powerful anticoagulant.[4]

Other actions of heparin include its having detoxifying effects on digitalis, cobra venom, and Congo red;[19] causing the clustering of chylomicrons and the packing of red blood cells after a heavy fat meal, resulting in a decreased sedimentation rate and hematocrit level;[61] inhibiting the action of hyaluronidase;[62] producing profound effects on low density lipoproteins in blood, resulting in low blood levels of these compounds and retarding the development of atherosclerosis in experimental animals;[24] and relieving markedly the pain due to angina pectoris far beyond its effective anticoagulant duration of activity.[24] No fibrinolytic activity for heparin has been proved.[9]

Heparin probably is released normally from the mast cells in anaphylactic shock,[23] during stress reactions,[5] and after irradiation,[64] resulting in a prolonged clotting time.

The action of heparin is effective in five to ten minutes following administration of moderate doses

and is dissipated completely within six hours.[30] The duration of the action is proportional to the dosage, but the compound is so effective that doses prolonging the action beyond six hours are dangerous in the first hours of administration.[30]

All toxic effects are due to extension of the physiologic actions of the drug. Large doses are not toxic, nor do they affect wound healing.[33] The only danger is that of hemorrhage or hematoma at the site of injection.[60] Some cases of severe sensitivity to heparin on first injection have been reported, but usually sensitization takes place gradually. Such reactions may be due either to the heparin or impurities. Test doses should be used on persons with allergic tendencies.[23]

Quantities as small as .1 micrograms of heparin will cause a slight increase in the prothrombin time.[4] Heparin achieved official status on November 1, 1950, with the USP unit described as the quantity of heparin sodium required to keep 1 cc. of plasma in a fluid state. The USP standard of potency is a minimum of 100 USP units per milligram.[17] Biologic methods of assay are necessary at the present time. The purified heparin sodium in 1 mg. quantities will prevent clotting for twenty-four hours in 500 ml. of cold cat's blood.

The determination of clotting time long has been accepted as the basic method for testing the action of heparin on the blood. However, determination of plasma antithrombin levels recently has been prescribed as a better test in this evaluation.[27] Dicumarol and Tromexan, other common anticoagulants, have no demonstrable effect on the blood antithrombin levels. It is said that only approximately 15 per cent of United States hospitals have laboratory facilities for the accurate testing of blood in patients receiving anticoagulant therapy.

Dependence on the body weight as a guide to dosage has been shown to be grossly inaccurate.[42] Test dosages should be given and coagulation time or some other suitable determination made to test the dose effectiveness. Usual therapeutic doses consist of 50 to 100 mg. injected intravenously every four to six hours.[48] Therapeutic increases in clotting time, which by the Lee-White method is five to ten minutes, are considered to be three to six times more than the normal level. The main disadvantage of using heparin lies in its cost, which is about $9 per day usually.

Sterile solutions of heparin sodium with varying strengths for intravenous use, sterile preparations with repository adjuvants (procaine and butacaine) to prolong the effect intramuscularly or subcutaneously,[44] and sterile preparations with vasoconstrictors and repository adjuvants combined still further to prolong effects of heparin are available commercially. The latter preparation may prolong the effect for as long as seventy hours.[54] The commercial products all are extracted from animal tissues.

The principal conditions in which this drug is contraindicated are subacute bacterial endocarditis, hemorrhagic diathesis, increased capillary fragility, purpura, bleeding lesions, and heparin sensitivity. It should never be used in cases which cannot be kept under careful observation for hemorrhage, or when clotting time determinations are not readily available.

Heparin is rapidly neutralized by protamine[45] in approximately equal amounts and by toluidine blue.[54] Aureomycin reduces the concentration of heparin in serum and increases the coagulability of the blood by some antiheparin activity.[64] Local anesthetics also will precipitate heparin.[39]

DICUMAROL

The story of Dicumarol began in North Dakota in the winter of 1919-1920 when cattle were dying of a mysterious hemorrhagic disease. Roderick and Schofield showed in 1928 that the etiologic agent of the disease was in the spoiled sweet clover ingested by the cows.[50] These men also showed that the prothrombin activity was decreased tremendously. Link and his associates undertook in 1933[56] to isolate the anticoagulant responsible for the disease. It was they who established that 12.5 per cent plasma was best suited to the study of drug action on the prothrombin time.

Campbell in 1938 isolated the first crystals of the compound which was identified in 1940 as 3,3′ methylenebis (4-hydroxycoumarin) (fig. 1). This discov-

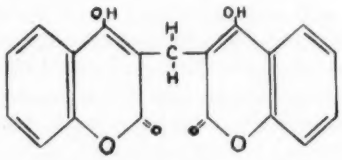

FIG. 1. Chemical structure of Dicumarol.

ery produced a relatively economical, effective anticoagulant which was destined to play an important part in anticoagulant therapy.

Dicumarol is absorbed from the gastrointestinal tract slowly, maximal absorption occurring at six hours after ingestion and complete absorption at twenty-four hours. The drug passes into the plasma, where it is bound to the plasma proteins, and its effect on prothrombin activity is first noted some eight to twenty-four hours after administration.

The effect of the drug lasts from twenty-four to forty-eight hours after the first decrease in prothrombin activity. Salicylic acid has been shown to be a degradation product of Dicumarol. This mechanism is obscure, but an enzyme system is thought to be

chiefly responsible. Other metabolic products of Dicumarol still are unknown, and all these are thought to be excreted through the urine and feces. Conflicting evidence exists as to the urine excretion of Dicumarol per se.[25] Hausner and others have demonstrated the molecule in the urine of rats. The molecule has been shown to be present in the liver, kidney, and lungs in the highest concentrations. The plasma Dicumarol concentration is higher than that of any tissue, although the unbound concentration is low.[56]

No certainty can be attached to the mode of action of Dicumarol. The fact is well known, however, that prothrombin concentration is diminished with Dicumarol administration. Quick believed this to be a competitive inhibition phenomenon in which Dicumarol replaces vitamin K as the prosthetic group in the enzyme responsible for the prothrombin production.[47] Another theory is that Dicumarol influences the production of altered prothrombin.[37]

In addition to its effect on prothrombin, Dicumarol also decreases the formation of accelerator globulin early in treatment;[20] increases or decreases fibrinogen level, depending on the dosage;[29] may act to inhibit the activity of obscure clotting factors; and in vivo can inhibit the adhesiveness of platelets.[65]

Most of the effects of Dicumarol overdosage are due to hemorrhage, with hematuria being the first sign of bleeding.

Dogs poisoned by Dicumarol showed, besides hemorrhage, toxic lesions of the small vessels, acute glomerular swelling, and toxic lymphoid tissue reaction.[15] It is to be emphasized that in man and animals no liver damage has ever been proved when only therapeutic doses have been given.

The responses to Dicumarol are far too varied to allow one to follow an empirical dosage chart.[56] Link said: "We never administer the anticoagulant without determining the prothrombin level... when I learn that a clinician has induced a frank case of hemorrhagic sweet clover disease of cattle in man, my usually buoyant spirits are bruised." Maintenance dosages are likewise determined from prothrombin levels. It is well to mention that the optimal prothrombin level sought in such treatment is still a center of tremendous controversy, and no definite statement regarding this will be made in this paper.

Dicumarol is prepared in capsules and in powdered form for oral administration.

The most important contraindication for its use is in liver disease. If damage is known to exist, therapy must be guarded. Active bleeding from any source is always a contraindication for anticoagulants in general.

Cases of subacute bacterial endocarditis usually are not treated with anticoagulants, since their use may prevent reinforcement of vascular wall injury by an organizing thrombus. Rupture of the vessel wall and perhaps fatal hemorrhage may result from such treatment.[56] Some avoid use of Dicumarol in renal disease, but as yet the evidence is inconclusive.

Vitamin K is the most useful antagonist of Dicumarol, since it may return blood prothrombin levels to a near normal level in twelve to twenty-four hours. There is considerable debate over the exact types of vitamin K most effective in combating hemorrhagic states caused by Dicumarol. Most types have proved effective, although massive doses may be required. The mechanism is probably that of displacement of Dicumarol in the prothrombin enzyme.[47] It is to be noted that vitamin K administration does not affect blood levels of Dicumarol.

Transfusion of whole blood is considered good therapy in combating Dicumarol overdosage.

Methyl xanthines antagonize the action of Dicumarol, though the mechanism is not clear.[52] When they are given with Dicumarol, the effect of the latter is strongly suppressed.[56]

COMPOUNDS RELATED TO DICUMAROL

Dicumarol has many related compounds, although none seem to be as effective as it is.

Phenylindandione has the advantage of reducing the prothrombin blood level for the first two or three hours after administration,[31] but this effect is only temporary; the main effect results some twenty-four hours after administration, and its similarity to Dicumarol coupled with some toxic side effects make this drug undesirable.

Compound 42 was studied by Link and his associates with the idea in mind that here might be a good anticoagulant. It was soon found to be an excellent rat killer because of its hemorrhagic phenomena. In fact, it is one of the best, since it is almost completely specific for rodents.

TROMEXAN

The compound known as Tromexan was synthesized by Rosicky by weakening the methylene linkage of Dicumarol and adding a carboxyl radical. This was done in an effort to improve the absorption of Dicumarol, assuming that the difficulty was in the splitting of the coumarin molecule. The result was a new anticoagulant having certain favorable properties.[14]

The structure for Tromexan is 3,3′-carboxymethylenebis (4-hydroxycoumarin) ethyl ester (fig. 2).

This compound is completely absorbed from the gastrointestinal tract in most persons.[12] The magnitude and time of the peak level of Tromexan in the plasma is variable, but is generally achieved in one to

six hours after administration. Dicumarol reaches its peak in twelve to thirty-six hours.[12, 14] Tromexan is not excreted in the urine, but several of its metabolic products are.[12, 14] Renal insufficiency still is a contraindication to Tromexan, even though the hyperreactivity in such subjects cannot be explained on the basis of decreased excretion.[12, 13] It is not extensively localized in tissues relative to plasma. The decline in the level of Tromexan in plasma after intravenous injection is rapid—about 25 per cent per hour. The

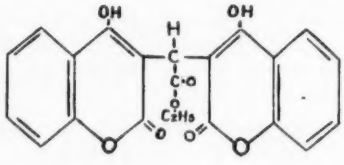

FIG. 2. Chemical structure of Tromexan.

Dicumarol level in plasma declines under the same circumstances at about 40 per cent per day. The actual rate, however, varies from individual to individual, but each person has a distinct pattern. As is true of both Tromexan and Dicumarol, the rate of decline in plasma level is inversely proportional to the dose; the larger the dose, the slower the decline in plasma level.[12]

The mode of action for Tromexan is essentially the same as for Dicumarol. There is little correlation between persistence of the drug in the body and peak prothrombin response. Peak plasma levels of Tromexan also are correlated poorly with prothrombin response.[12] Giving repeated doses of it will cause a cumulative effect on the prothrombin level, even though the drug remains in the plasma for only a few hours after administration. Dicumarol still has a high plasma level when the prothrombin time begins to respond.[12] On discontinuation of Tromexan therapy, an average of one to two days elapse before the prothrombin time returns to normal. With Dicumarol, this time is about four days.[12, 14, 58] Clinical trials have shown that when comparable prothrombin levels are maintained, Tromexan is as effective as Dicumarol.[13, 14, 63]

This compound is one-fifth as potent as Dicumarol, but has only one-twentieth the toxicity. Vitamin K and transfusions are used to combat overdosage, as with Dicumarol.

Tromexan therapy is difficult to control because of the rapid and sometimes unpredictable changes in prothrombin time, even when regular daily doses are given. In general, however, a daily determination of prothrombin time will reflect the effect of the previous day's medication, so that dosage may be altered accordingly.[12, 13, 14, 58] These dosages may vary with the individual patient. Split doses have been found to avoid much of the fluctuation in prothrombin levels.[12, 13, 14, 58] In general, an initial dose of 900 to 1,200 mg. is given, followed by a daily maintenance dose of 300 to 600 mg. The drug is packaged as uncoated scored tablets of the pure compound.

Grossly, Tromexan has the same indications, contraindications, antagonism, and method of administration as Dicumarol.

SALICYLATES

In early reports salicylates were described as the etiologic agent of hemorrhagic phenomena in salicylate-treated patients.[18] Investigation has shown them to be degradation products of Dicumarol, and this has prompted further work in the field.[6] The mechanism of action centers around the reduction of prothrombin in the blood.[19] Field has shown that, as in Dicumarol antagonism, vitamin K will raise the blood prothrombin level in salicylate overdosage.[22] It is now standard treatment to give the vitamin in all cases in which large doses of salicylates are used. In many countries where malnutrition and starvation are rampant, cases of hemorrhagic disease due to salicylate ingestion are commonplace, and in these places particularly, vitamin K is indicated.

The mechanism of action is still unknown and consequently is a center of controversy. Field, Spero, Link, and others[22] stressed their belief that salicylates act only when vitamin K function is impaired, and that these agents have a low affinity, compared to Dicumarol, for the site of vitamin K action. However, the evidence is inconclusive, and until further proof is gathered, salicylate action will remain a mystery.

ALPHA-TOCOPHEROL

The action of alpha-tocopherol (vitamin E) on coagulation is interesting, but of no practical value in man as yet. Three main effects have been demonstrated: (1) It acts as a vitamin K–like substance, since vitamin E deficiencies cause hemorrhagic states in experimental animals.[54] (2) It acts as an antithrombin, preventing the formation of fibrin from fibrinogen.[21] (3) It has been shown to have antithromboplastic activity.[36] Since these actions have been demonstrated only experimentally in laboratory animals, the use of vitamin E as a therapeutic agent in man is yet to be determined.

SULFONAMIDES

Sulfonamides affect prothrombin levels of the blood much the same as Dicumarol. However, their action is thought to be on the bacteria in the gastrointestinal tract, which play an important role in vita-

min K synthesis.[11] Administration of vitamin K in conjunction with sulfonamidex therapy will decrease the likelihood of hemorrhage.

ANESTHETICS

Anesthetics like chloroform are notorious for causing liver damage, which results in reduction of blood prothrombin and fibrinogen levels. Recovery usually occurs in about a week, with both levels returning to normal.

TRYPSIN

A new and possibly revolutionary drug has now come into limited use. This is trypsin. First crystallized by Northrup, it is a protein with a molecular weight of 35,000, containing aspartic and glutamic acids, arginine, and lysine. The Armour Laboratories have perfected a highly purified crystalline trypsin called Enzar, upon which many studies have been made.

$L.D._{50}$ studies have shown a tremendous species difference; for example, $L.D._{50}$ for guinea pigs is 80,000 Armour units per kilogram of body weight; for mice 40,000 Armour units per kilogram; for rabbits 5,000 Armour units per kilogram; and for cats 4,000 Armour units per kilogram. These studies were based on rapid infusion and high drug concentration. When trypsin was administered in a concentration of 3,000 Armour units per cubic centimeter at a rate of 6 to 10 drops per minute, rabbits tolerated 250,000 units and dogs, 1,250,000 units with no ill effects. It is evident that concentration and rate of infusion are keys to the problems of administration.

A circulating trypsin inhibitor has been found to combine with 97 to 99 per cent of the trypsin to form a stable, irreversible compound. Some 1 to 3 per cent of the trypsin remains free to act. However, the action of the trypsin inhibitor is depleted accordingly by the chemical combination, and this may be of some clinical importance. In such diseases as pneumonia, typhoid fever, rheumatic fever, tuberculosis, hyperthyroidism, malignancies, and anemias, trypsin inhibitor levels, along with fibrinogen levels, rise. Since Enzar reduces these trypsin inhibitor and fibrinogen levels, it seems that administration of Enzar provides a method which alters the natural history of a disease.

The specific action of trypsin is based on the hydrolysis of circulating proteins and their products—fibrin, prothrombin, fibrinogen, thrombin, globulin, and albumin in that order. Thus, in the presence of a fibrinous clot, trypsin acts to dissolve the clot. If no fibrin is present, 300 units of the drug in 20 cc. of blood will result in clotting; but if 400 units are added to another 20 cc. of blood, the latter will be incoagulable. At first this would seem to be an almost hopeless dilemma, but upon closer inspection, it is seen that all available trypsin was utilized in the first tube to convert prothrombin to thrombin. In the second 20 cc., there was an excess of the trypsin, and this caused fibrinogenolysis. Thus no clotting occurred. Had fibrin been present originally, the drug would have acted to form a soluble polypeptide from the fibrin, with digestion of the clot resulting. It is well to note that extreme caution has to be used in administering this drug, since small changes in concentration cause such varying results.

A problem is posed by the formation of the polypeptide from the fibrin. Increased vascular permeability and, perhaps, some intestinal spasm result. Because antihistamines decrease vascular permeability, it has been postulated that they should be used along with trypsin.

Trypsin also activates fibrinolysins by conversion of profibrinolysins, but one of the most important actions is on the individual blood cell. Tryptic action on surface protein elements causes increased volume, increased mechanical fragility, and increased osmotic fragility of the red cell. Also, the red cell becomes agglutinable, but the presence of a reverser substance, for which the cell manifests the stronger affinity, erases this as a clinical problem. All these actions on the cell affect the oxygen carrying capacity in such a way as to contraindicate the drug in congestive failure. Cyanosis also has been reported, and it has been explained on a basis of methemoglobinemia. Leukocytes are affected by action on surface proteins also.

The study of trypsin is far advanced in experimental animals, but the greatest test of all, the one on man, has not been adequately made. Until this drug has been used thousands of times with excellent results, judgment must be reserved.

SUMMARY

This paper has served to acquaint the reader with a knowledge of anticoagulants, their uses, and their successes. But it is to be stressed that they are no panacea for thromboembolic disease. There are those who would say anticoagulants are the answer; there are others who would as dogmatically state they are useless. Between these two lies the investigator who would be as the politician and tread the middle of the road. Herein may lie the most ideal approach to this drug therapy. Anticoagulants have been used extensively, but they are still young as a group and still growing in number. The pharmacologist and the clinician will have to study this problem a great deal further before a definite conclusion can be reached.

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Experiences with Long-Term Anticoagulant Therapy

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THE value of anticoagulant therapy in thromboembolism has been proved, without doubt, by numerous investigators in this country and abroad. The treatment has been confined mostly to the acute thrombotic diseases and embolic complications. However, there is a large group of patients in whom the danger of thromboembolism may be present for a period of months, years, and even a lifetime. In such cases the prolonged use of anticoagulants appears logical as a prophylactic measure. The general recognition of the value of such treatment has been greatly hindered by fear of their toxicity, which is unwarranted because the untoward reactions encountered are due more to mismanagement than to the dangerous properties of the drugs. The literature discussing the value of prolonged anticoagulant treatment is sparse; however, Wright,[6] Nichol,[4] and Shapiro[7] have reported favorably on this subject.

A similar opinion was expressed by us in a previous publication.[3] Since that time our group has been expanded to include 78 patients who have now been observed over a period of up to five years. It is the purpose of this presentation to elaborate on our further experiences with such therapy.

The minimum of two months' treatment was arbitrarily selected for the inclusion of cases in this group. During the time of the study, a considerable number of persons received anticoagulants for a lesser period; they either were considered to be unsuitable or refused such prolonged therapy.

METHODS AND COMPLICATIONS

Successful and safe long-term anticoagulant therapy first must entail certain basic principles: (1) a trustworthy patient and family who will follow explicitly directions as to dosage and as to the need to observe carefully the early signs of intoxication should they develop; (2) a reliable laboratory with well trained technicians who are able to perform the prothrombin test with a minimum of error; (3) a physician who understands thoroughly the indications, contraindications, dosage, and toxicity of the drugs. Although hospitalization is desirable for acutely and seriously ill patients, it was not considered necessary in our average case. In many persons the treatment was started while they were at home; the majority were entirely ambulatory from the onset. Practically all patients of our series were able to continue their usual activity whether it be a trade, a profession, or household duties.

At the beginning of the anticoagulant therapy a graphic chart was prepared for each patient and continued during the entire time of his observation. This chart showed the prothrombin time in seconds, the percentage of prothrombin depression, and the daily dose of anticoagulant administered. The Link-Shapiro modification of the Quick prothrombin method was used in this study. Special precautions were taken to standardize the technique and to eliminate errors as much as possible. Each new batch of thromboplastin (Simplastin*) was compared carefully with the previous one to assure uniformity of action. Little variations from the normal standards were encountered during the entire time of observation. The normal prothrombin time ranged from 13 to 15 seconds. The identical method of prothrombin determination was used for hospitalized patients to make the transition to home care safe and simple. Blood tests initially were performed daily, then every three to four days, and after a short time once a week. Finally, when the patient's prothrombin level had become stabilized, the tests were done only every two weeks and occasionally even at longer intervals, but usually the two week schedule was followed rigidly.

The ideal drug for prolonged anticoagulant treatment should be administered orally, be well tolerated by the gastrointestinal tract, be without toxic effect on the parenchymal organs, depress the prothrombin level evenly for prolonged periods, be easily neutralized by available antidotes, and be relatively inexpensive. Heparin and its derivatives are not suitable for prolonged anticoagulant therapy for obvious reasons. Tromexan also is not too useful for this purpose because of its transient effect which causes wide swings of the prothrombin time and necessitates frequent blood tests. Phenylindandione resembles Tromexan in its action but the effect is more prolonged. Both Tromexan and phenylindandione should be given twice daily for their optimum action. This is more cumbersome than Dicumarol and Cumopyran administration, which requires only daily dosage. Dicumarol still appears to us at this time to be the drug of choice for prolonged anticoagulant therapy. Cumopyran is almost equally useful for this purpose; however, the prothrombin depression is more difficult to maintain and the effective dosage is more variable. Therefore, the drug requires more careful supervision, especially during the initial period of treatment. It has been used only when patients became resistant to Dicumarol.

*Chilcott Laboratories.

There is no need for indiscriminate change from one anticoagulant preparation to another. It is much safer to restrict oneself to one or two drugs and to prescribe them only in one size tablet or capsule. In our group of patients, 50 mg. of Dicumarol or 25 mg. of Cumopyran tablets were used exclusively.

Our graphic chart proved helpful for the calculation of the dosage. Immediately after each prothrombin test the total dosage for the following period was calculated on the basis of the previous amount administered, the change of the prothrombin time, and the tendency to further prothrombin depression or elevation; this quantity was then evenly divided into daily doses. The patient was instructed to write the number of tablets or capsules prescribed for each day on a calendar and to mark it off after taking the drug. The total dosage of Dicumarol varied from 0 to 100 mg. daily and 200 to 700 mg. weekly, and that of Cumopyran from 0 to 75 mg. daily and 100 to 500 mg. weekly. So far none of the patients has become resistant to both drugs. In case this should happen a shift to phenylindandione probably would be attempted. It is usually stated that menstruation, febrile diseases, and various drugs such as aspirin, digitalis, and antibiotics influence the prothrombin time, necessitating a change of anticoagulant dosage. This has not been brought out in our series. Fluctuation of the prothrombin level does occur for obscure reasons but is not related to the above mentioned factors.

Usually the earliest sign of anticoagulant toxicity is microscopic hematuria, followed later by gross bleeding from the kidneys and by subcutaneous hemorrhages. Gastrointestinal, respiratory, and intracranial hemorrhages have been reported in severe prothrombin depression. However, in the presence of coexisting disease, such as peptic ulcer, diverticulosis, coryza, bronchiectasis, tuberculosis, and encephalomalacia, the former complications may occur even with small doses of anticoagulants. We have observed several cases of epistaxis and hemoptysis in the presence of infections of the upper respiratory tract. One patient with a history of a traumatic olecranon bursitis developed a hemorrhagic effusion in this bursa. Another patient, with an endocervical carcinoma, experienced metrorrhagia. From such observations the contraindications to anticoagulant therapy is self-evident. In addition to various hemorrhagic states, such as occur in hepatic and renal disturbances and in blood dyscrasias, the existence of any lesion that may bleed is a contraindication to such therapy. However, under certain circumstances these toxic effects will have to be balanced against the dangers of thromboembolism.

The bleeding tendency is best counteracted by vitamin K_1 (Mephyton*) given in a dose of 25 to 50 mg. intravenously and repeated as indicated by the prothrombin level. Other vitamin K preparations are relatively ineffective. Modest dosage of vitamin K_1 is advisable to prevent the shortening of the prothrombin time below the pretreatment value. This has occurred in our experience contrary to previous reports denying this possibility. Smaller doses of vitamin K_1 also will avoid the development of refractoriness to anticoagulants. Blood transfusions are ordinarily not needed since the introduction of vitamin K_1.

Apart from the hemorrhagic manifestations the toxic symptoms of Dicumarol and Cumopyran have been mild. There were no adverse effects upon the hematopoietic, hepatic, and renal functions. A future communication will deal with these in greater detail. Individual doses of Dicumarol exceeding 150 mg. sometimes produced nausea, indigestion, and lightheadedness. For this reason the drug was administered daily in small amounts instead of every few days in larger quantities as recommended by Shapiro.[8] In none of our cases did the drug have to be omitted or reduced because of such effects.

INDICATIONS

Not considering rare disorders such as essential thrombophilia, hepatic vein thrombosis (Budd-Chiari), thrombotic obstruction of the abdominal aorta (Leriche), and pulmonary hypertension due to recurrent embolization (Ayerza's syndrome), prolonged anticoagulant therapy should be considered in three groups of diseases: (1) chronic, recurrent, and migratory peripheral thrombophlebitis; (2) cardiac diseases with intracardiac, coronary, pulmonary, and systemic thromboembolism; and (3) cerebrovascular thromboembolism.

Peripheral Thrombophlebitis.—One of the most common complications of posttraumatic, postoperative, and postpartum states as well as medical disorders requiring prolonged bed rest is peripheral phlebothrombosis and thrombophlebitis with or without pulmonary embolization. It has been stated by Wright[9] that "patients who have had one pulmonary embolus, if untreated, have a twenty per cent chance of death from subsequent pulmonary emboli." Therefore, anticoagulants are generally accepted as the treatment of choice should such phenomena occur. However, there is no agreement about the duration of such therapy. Unfortunately, no clinical criteria and laboratory tests are available that would indicate when the danger period has passed. For this reason it seems advisable to give the drugs for several months instead of a few weeks, especially in persons with persisting obstruction of the venous blood flow or in those with disorders favoring blood coagulation. It is also possible

*Merck & Co.

that such therapy will contribute materially to the complete restitution of the normal circulation because it may hasten the disintegration of the blood clot.[9] If thrombophlebitis recurs or migrates, there is certainly an indication for the long-time administration of the anticoagulant drugs provided that Buerger's disease, malignancy, and blood dyscrasias are not present. In the latter conditions the drugs are useless and perhaps even contraindicated. Finally, there is a group of persons with peripheral edema, varicose veins, ulcers, and eczema who are considered to have residual venous insufficiency while actually having a persistence or recurrence of the thromboembolic disease. Such persons complain of pains and cramps in the legs, spells of fever, cough, and expectoration. Clinical examination may show localized phlebitis, recent or past pulmonary infarctions, accelerated blood sedimentation rate, and elevated white blood cell count. In such cases the prolonged anticoagulant therapy will have the most gratifying effect.

Cardiac Disease with Thromboembolism.—The occurrence of intracardiac, pulmonary, and peripheral thromboembolism constitutes one of the serious complications of various heart diseases mostly in the presence of congestive heart failure. Fortunately, this does not occur frequently enough to warrant routine anticoagulant treatment; however, under certain conditions, for example in mitral stenosis with auricular fibrillation, the incidence of clotting phenomena and embolism is great, especially if one episode already has occurred. It has been estimated that the recurrence rate after two attacks of embolism is almost 100 per cent.[10] Under such circumstances the introduction and maintenance of a hypocoagulable state is indicated as long as such a danger period persists.

The value of anticoagulants in acute myocardial infarction is firmly established by the reports of the Committee on Anticoagulants of the American Heart Association. In our series, Dicumarol therapy, once started, was continued indefinitely, whenever possible, with the purpose of lessening the chances of future coronary thrombosis. Such prolonged treatment is especially justified if a history of previous coronary occlusion or pulmonary or systemic embolization exists, or if angina pectoris, congestive heart failure, or cardiac aneurysm develop after the acute attack.

The use of prolonged anticoagulant therapy in selected cases of coronary insufficiency, although not universally accepted, has proved to be of definite value in our estimation. This opinion is shared by other groups of investigators.[4, 6, 7] Such treatment has been given only to patients whose anginal attacks were severe, sustained, or frequent or had other characteristics of impending infarction. A decrease or disappearance of pain was observed in practically all cases following this therapy, even in those who previously had failed to respond to the conventional drugs. The mechanism of such action of the anticoagulants is not clearly understood. A vasodilating property and a promotion of the coronary blood flow has been suggested.[5] The treatment in some patients has been maintained continuously for forty months without any deleterious effect, nor has anyone in this group developed an acute myocardial infarction.

Cerebrovascular Thromboembolism.—Apart from the prevention of cerebral emboli, following various cardiac diseases, anticoagulants are useful in certain cases of cerebrovascular thrombosis. In recent years the diagnosis of chronic progressive occlusion of the carotid, vertebral, and basilar arteries has been made more frequently on the basis of clinical signs, symptoms, and angiography.[1] In such cases the use of Dicumarol and related drugs seems to be logical. The acute cerebrovascular accident so common in clinical medicine is frequently due to a thrombus involving one of the smaller vessels of the brain. The use of anticoagulants in this condition must be introduced only after the presence of cerebral hemorrhage is ruled out. Unfortunately, the distinction between the two entities is extremely difficult, irrespective of the various symptoms and signs generally mentioned in the differential diagnosis. Therefore, the use of Dicumarol in cerebrovascular diseases carries a greater risk than in other vascular occlusions. However, anticoagulants have shown gratifying results in patients with recurrent attacks of cerebrovascular thrombosis.

RESULTS

The results of prolonged anticoagulant therapy are difficult to evaluate because of the great variations in the course of thromboembolic diseases. Furthermore, this treatment requires close medical supervision of the patient which might in itself be a factor improving his prognosis. Nevertheless, the following results appear convincing as to the efficacy of such therapy.

Seventy-eight patients received anticoagulants over a period of two to sixty months totaling 823 months, with an average of ten months per person. There were 23 cases of coronary thrombosis, most of which were recurrent or complicated. The period of treatment varied from two to thirty-nine months and averaged thirteen months. In this group there were 2 failures. One patient, as mentioned in a previous communication,[3] sustained a fatal recurrence during the time of a 25 per cent prothrombin depression. Another, a 65 year old white man, was placed on Dicumarol after his third heart attack and had been asymptomatic for a period of twenty-two months when cortisone was administered because of a severe shoulder-hand syndrome. A fatal coronary thrombosis followed within a few

days even though his prothrombin level was within a therapeutic range. This death, perhaps, can be attributed to the steroid hormones, which may produce a hypocoagulability even in the presence of a prothrombin deficiency.[2]

Eighteen patients with recurrent cerebral thrombosis and 3 with multiple cerebral emboli received treatment from two to sixty months and averaged twelve months. None experienced any further episodes during treatment; 4 had recurrences after cessation of therapy.

In 18 patients with chronic, recurrent, or migratory peripheral thrombophlebitis the results were uniformly good. Duration of treatment varied from two to fourteen months but was usually limited to four months.

Eight cases of congestive heart failure with pulmonary and systemic emboli were treated with anticoagulants for two to forty months and averaged twenty-two months. No vascular episodes recurred during the time of treatment.

There were 8 patients with severe coronary insufficiency who received the drugs from two to forty months and averaged twelve. All experienced partial or complete relief of angina, many exhibited electrocardiographic improvements, and none sustained a coronary thrombosis during the time of treatment.

SUMMARY

The experiences of prolonged anticoagulant therapy in a group of 78 patients have been described. The simplicity and safety of such treatment in ambulatory cases has been demonstrated. The efficiency of such drugs in the treatment and prevention of various thromboembolic diseases has been evaluated.

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THE SURGICAL TREATMENT OF CARDIOSPASM
Preliminary Report

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THE term "cardiospasm" has been used frequently to indicate a benign obstructive lesion at or near the junction of the esophagus with the stomach, usually associated with marked enlargement of the esophagus. Probably because the cause is obscure, there has been disagreement concerning the name which best describes the condition. In 1940 Ochsner and DeBakey[12] published an extensive review of the literature and concluded that the term "achalasia" is preferable. The word "mega-esophagus" was used by Wangensteen in the title of his recent paper,[14] but in subsequent discussion he suggested that "dystonia" is better. Fortunately, in spite of disagreement regarding the etiology and a name for the lesion, there is at this time a satisfactory crystallization of opinion about definitive surgical treatment. A satisfactory operation for cardiospasm has become available and recognized none too soon, because many of the surgical procedures carried out during the past five years have been followed by serious complications.

From the Division of General Surgery of the Henry Ford Hospital. Read before the Section on Surgery, Texas Medical Association, Annual Session, Houston, April 29, 1953.

UNSATISFACTORY TECHNIQUES

With the advent of good anesthesia for thoracic operations and the availability of antibiotic drugs, it became common practice to open the lumen of the esophagus in the lower part of the chest and to carry out various surgical procedures on the esophagus and the stomach. The most common indication was malignancy of the cardiac end of the stomach or the esophagus. The results of this type of surgical intervention were encouraging, and it is not surprising that thoracic surgeons had no fear of dealing with the ap-

parently simpler condition of a benign stricture involving perhaps less than a centimeter of the lower esophagus. Obviously, the constricted lumen could be enlarged by making a longitudinal incision and closing it transversely. Such an operation was first carried out by Wendel[15] in 1910. I performed this operation on 3 patients in 1946 and was immensely pleased with the immediate results. Dysphagia was relieved, and within a few weeks the size of the esophagus in each case was reduced to almost normal proportions (fig. 1). During the next four years, I operated on 6 other patients in a similar manner. In 1948 Kay[8] reported that he had done this operation in 17 cases and was pleased with the results.

In 1912 Heyrovsky[7] proposed an anastomosis between the fundus of the stomach and the dilated esophagus to by-pass the stricture, and his operation was subsequently modified by Gröndahl,[5] who described a cardioplasty which was analogous to the plastic operation at the pylorus performed by Finney. The latter method was used in a number of cases by Ochsner and DeBakey,[13] Bell,[1] and Grimson and his co-workers.[4]

As the years passed, it became evident that something was wrong with these operations which made a generous opening between the esophagus and stomach with destruction of the sphincter mechanism. Five of my 9 patients who had had the Wendel cardioplasty returned with evidence that the reflux of gastric juice into the esophagus had done considerable damage. All had definite signs of esophagitis and several had severe bleeding from ulceration. The following case report is instructive.

B. B., a 70 year old white woman, was observed in February, 1951. For eleven years she had had dysphagia which had been only slightly and temporarily relieved by dilatation. The esophagus was huge. A Wendel cardioplasty was carried out, and convalescence in the immediate postoperative period was uneventful. The patient was discharged two weeks after the operation but was readmitted the next day with evidence of massive gastrointestinal bleeding. During the next two years she received approximately fifty transfusions. Resection of the lower esophagus and acid-bearing portion of the stomach was advised, and this procedure was done by a surgeon in another city.

The description by the pathologist (Dr. Alfred E. Rhodes) of the specimen was as follows:

"The specimen consisted of a tubelike segment of the lower esophagus with the adjacent portion of the stomach. The esophageal portion was edematous. The lining showed an irregular superficial ulceration 2 cm. in its largest diameter at the level of the cardia. The base and edges of the ulcer showed grayish-white tissue. The lining of the esophagus showed numerous patches of white, thickened tissue.

"*Microscopic Examination:* Sections showed a portion of the esophageal and stomach wall partially covered by stratified epithelium and stomach mucosa. Proximal to the stratified squamous epithelium for some distance the whole esophageal lining was necrotic as was also the underlying tissue, which contained many fragmented leukocytes and deeper, round cell and granulocytic infiltration. Much of the connective tissue and muscular wall of the gastric portion of this section showed hyalinization.

Diagnosis: Necrotic and chronic fibrosing esophagitis with degenerative changes in the wall of the cardiac portion of the stomach."

It became obvious that one could not apply the experience with partial gastric resection and esophagogastrostomy to the management of cardiospasm. Such operations for cancer were accompanied not only by an automatic bilateral vagotomy but also by a resection of all or most of the acid-secreting portion of the stomach. Reflux esophagitis was absent or minimal, and in any event, the patient could be told that he was better off with the substernal discomfort than to be dead of cancer. The addition of vagotomy to the operation of cardioplasty for cardiospasm appeared to offer some promise of ameliorating the peptic esophagitis, but Kay[9] informed me that he had tried it and observed it to be unsatisfactory. Wangensteen[14] tried a radical procedure, consisting of resection of the lower end of the esophagus and most of the stomach with end-to-end anastomosis and a pyloroplasty to favor emptying after the concomitant vagotomy. His patients were satisfied, but at the time of presentation of his paper, Wangensteen was inclining toward the more conservative procedure about to be described.

HELLER PROCEDURE

As early as 1913 the German surgeon Heller[11] had described his operation of extramucosal cardioplasty. This was a Ramstedt procedure at the cardiac rather than the pyloric end of the stomach. Heller made two longitudinal incisions in the muscular layers of the stomach and esophagus, on the anterior and posterior aspects of the cardia (fig. 2). Heller told how from the first day the patient could eat bread, meat, and potatoes; previously, he had been able to ingest fluids only, had a foul breath, and was losing weight.

It is difficult to understand why this brilliant result did not cause the operation to become generally popular. As a matter of fact, it was used considerably, since Ochsner and DeBakey[12] in 1940 were able to collect 104 instances of Heller operations. Recurrence of symptoms took place in 14 cases, and there were 4 deaths. Two of these deaths were due to infection which followed tearing of the mucosa, 1 to perforation following subsequent dilatation, and 1 to pulmonary embolism. Considering that all or most of these operations were done through the poor exposure obtained by the abdominal approach and all were done in the preantibiotic era, the record is far from black. A little later the British surgeon Maingot[11] reported using Heller's operation in 39 cases with 37 excellent results.

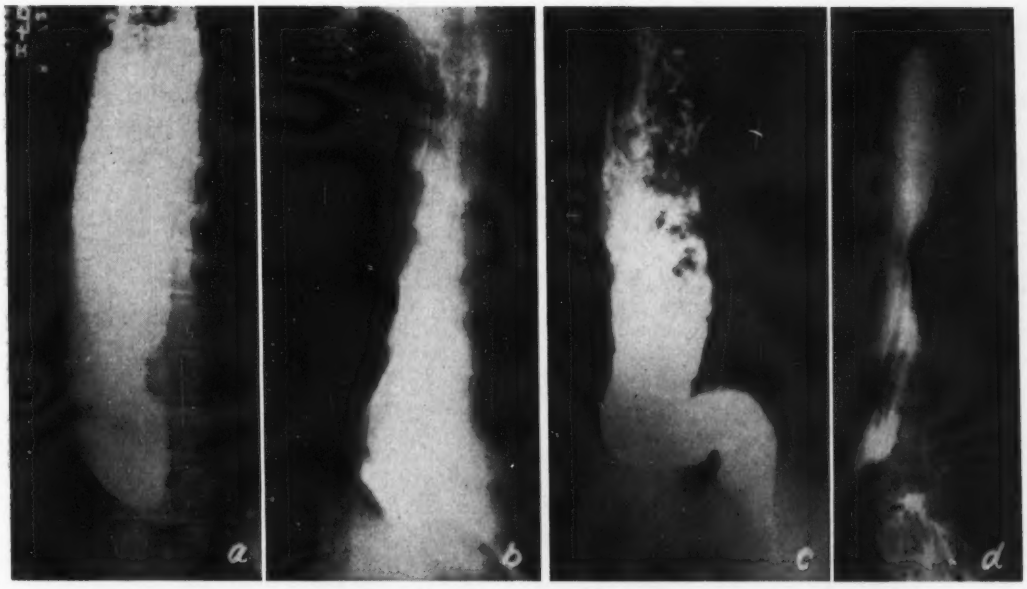

FIG. 1a and b. Roentgenograms of the esophagus showing marked dilatation due to cardiospasm before operation (*a*) and reduction in size only twelve days after a cardioplasty of the Wendel type.

c and d. Roentgenograms of the esophagus made before operation and nineteen days postoperatively showing relief from cardiospasm obtained by the Wendel operation.

Nevertheless, in 1951 at the meeting of the American Association for Thoracic Surgery, DeBakey[2] discussed Kay's paper[10] on achalasia of the esophagus as follows: "The fact that this operation was proposed by Gottstein approximately fifty years ago, first performed by Heller almost forty years ago, subsequently modified by a number of other surgeons, and abandoned and revived from time to time, strikes me as significant. Such considerations in the historical development of an operative procedure usually reflect dissatisfaction with its ultimate results as well as with its rationale. For these reasons, it would seem desirable in the current revival of this operative procedure to provide additional facts for its rationale. The question which needs to be answered is why is it a better operation now?"

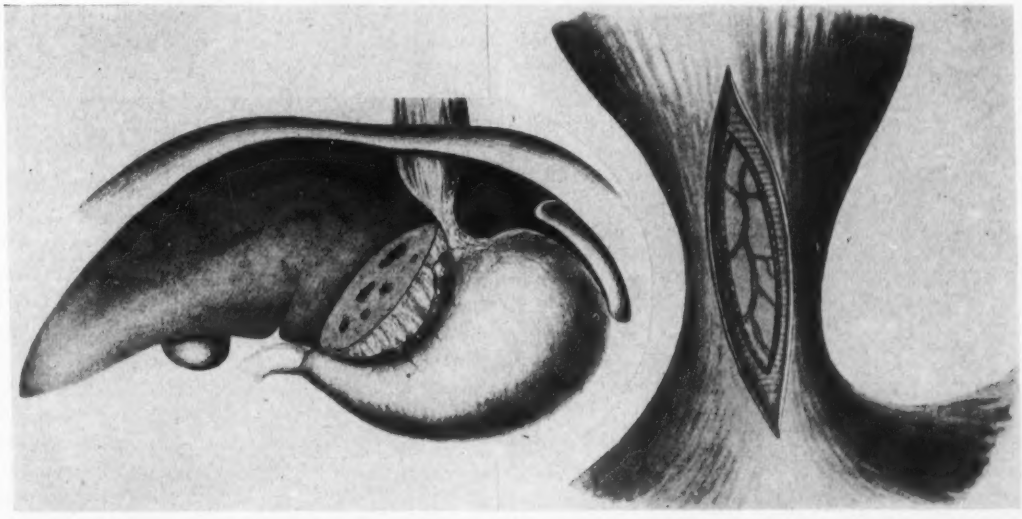

FIG. 2. Copies of the illustrations from Heller's article on cardiomyotomy[6] showing theoretical exposure by the transabdominal approach and the two extramucosal incisions into the esophageal and gastric walls.

After discussing my experience with the Heller operation, I shall try to answer Dr. DeBakey's question.

I have performed Heller's operation on 9 patients. The technique has been as follows. Through a posterolateral incision, the left thoracic cavity is entered through the eighth interspace (fig. 3). The eighth rib is transected posterior to its angle. The pleura

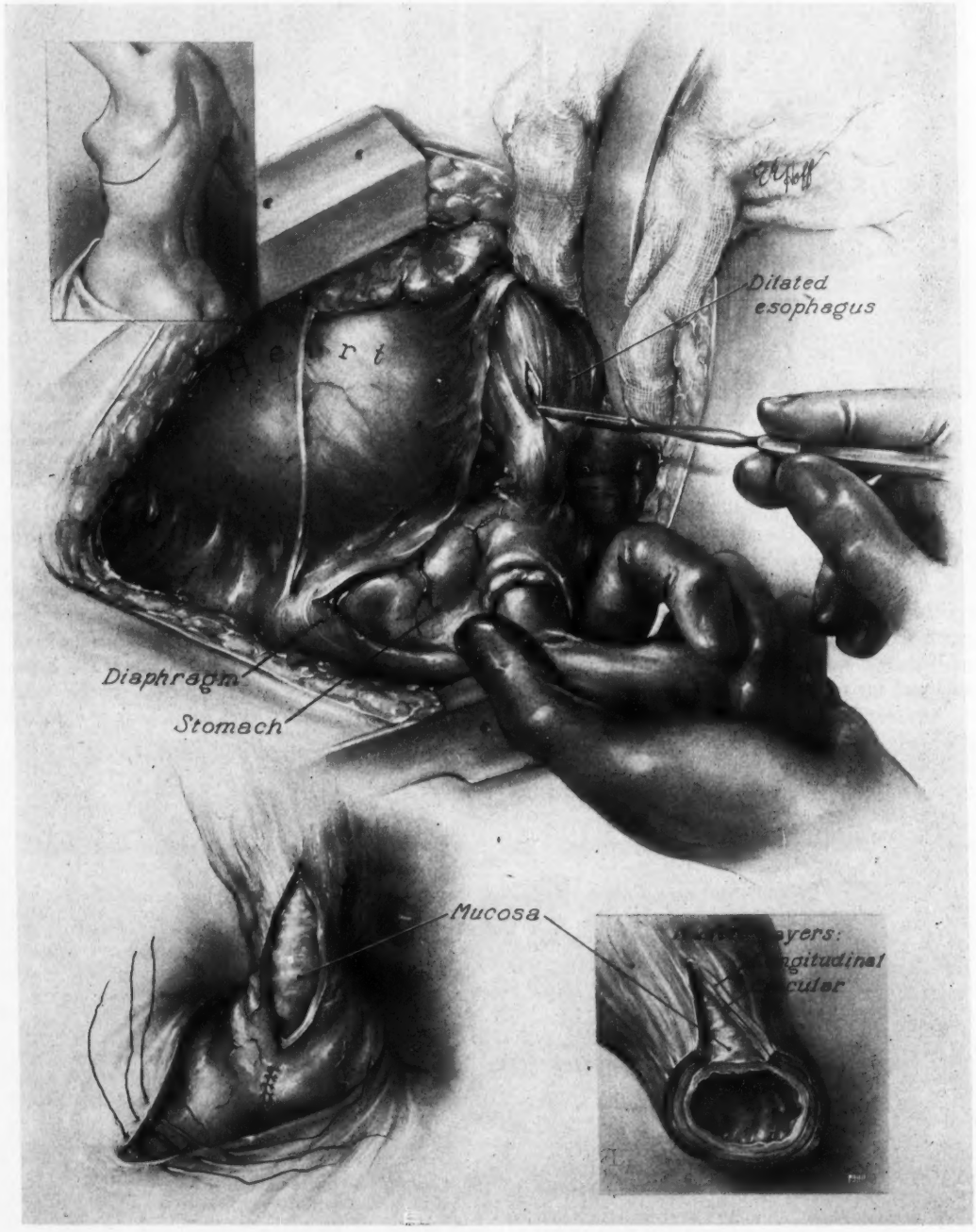

FIG. 3. Diagrams illustrating the technique of cardiomyoplasty, modified Heller method.

overlying the lower part of the dilated esophagus is divided and reflected. A short incision is made in the hiatus of the diaphragm to permit visualization of a portion of the stomach. The proposed line of the longitudinal incision, measuring between 4 and 5 inches in length, is cleared of areolar tissue and vascular channels. Beyond the proposed incision in the muscular coats of the stomach, a small incision is made into the lumen of the stomach through which the left forefinger is inserted. This finger explores the area of stricture and is passed through it if possible. With the finger as a guide to prevent injury to the mucosa, the serous and muscular coats of the stomach and the muscular wall of the esophagus are incised. The finger immediately appreciates the relaxation which has been produced. The opening into the stomach is then closed, the incision in the hiatus is repaired, and the chest is closed without drainage after the instillation of penicillin. The patient is fed intravenously for a few days, during which time the bare mucosa should have become agglutinated to the adjacent structures. Nausea and vomiting during the immedate postoperative period might result in rupture of the esophagus at the operative site.

There has been no mortality or important morbidity attributable to the operation. In 1 patient the operation was done for palliation in the presence of complicating carcinoma of the middle portion of the esophagus.[3] The subsequent autopsy in this patient, who died of perforation of the carcinoma into both main bronchi, gave an excellent opportunity for examination of the adequacy of the Heller procedure. The obstruction had been completely relieved.

The other 8 patients have had gratifying clinical results (fig. 4) and several have been followed for more than a year.

In answer to Dr. DeBakey's question, "Why is the Heller operation better now?" one might first say that it was never as bad as implied, since, it will be recalled, there were only 14 recurrences of symptoms and 4 deaths in 104 collected cases. It is likely that the thoracic approach leads to a more thorough operation, since the incision through the esophageal musculature can be made adequately high. Furthermore, all of the factors which make modern thoracic surgery safer are operating to make the patient receiving the Heller operation today surer of leaving the hospital without misfortune.

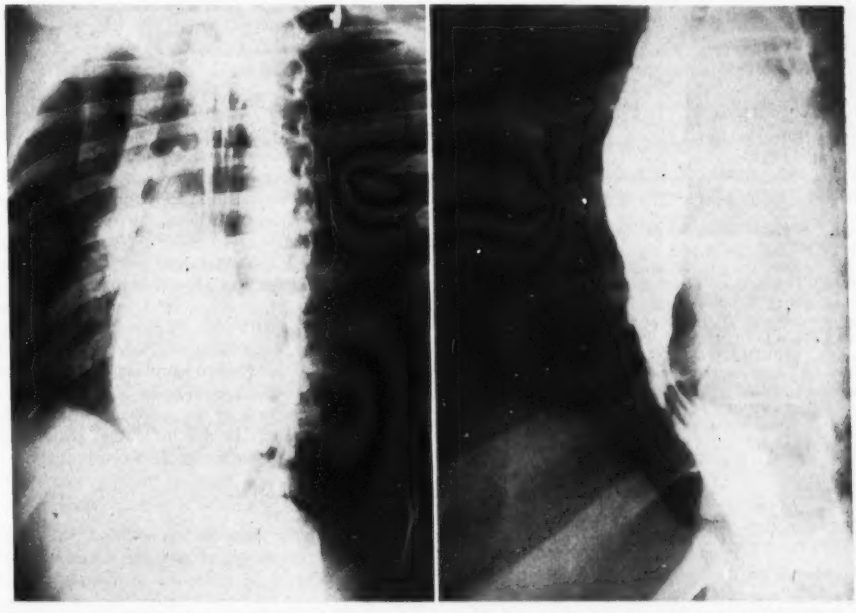

FIG. 4. Roentgenograms made before operation and three months postoperatively showing cardiospasm treated by the Heller operation.

HYDROSTATIC DILATATION

Finally, a word should be said regarding hydrostatic dilatation. It is probable that gastroenterologists and endoscopists will continue to give relief to a certain number of patients with cardiospasm for some time to come. However, as they note recurrences of strictures and find their patients drifting off to other clinics, it is likely that they will recommend definitive surgical

correction (the Heller operation) with increasing frequency.

SUMMARY

Personal experience with 18 cases of cardiospasm (mega-esophagus, achalasia, esophageal dystonia) has been presented. Cardioplasties of the Wendel type were done in 9 cases with unsatisfactory end results because of the incidence of esophagitis and peptic ulceration. Extramucosal cardiomyotomy of the type proposed by Gottstein and first performed by Heller has been carried out in 9 cases with gratifying results.

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ABSTRACT OF DISCUSSION

DR. H. T. BARKLEY, Houston: I am happy that the problem of cardiospasm is being brought to our attention. In this connection I cannot resist the temptation to say something about the role of emotional disturbance in etiology and stress the importance of the psychosomatic approach. The patient probably will develop other gastrointestinal symptoms unless the psychosomatic factor is considered.

In my opinion hydrostatic dilatation does not cure cardiospasm; I have seen the condition worsened by dilatation. At the same time, cardioplasty is not without hazards. I have been impressed with the thickening of the muscularis at the cardia causing a constricting ring. Extreme mega-esophagus must be resected. It appears that the Heller procedure offers the safest relief of cardiospasm and its symptoms.

Recently I have seen a patient with cardiospasm and carcinoma, indicating the need for endoscopic examination of all patients with cardiospasm before any type of therapy is considered.

DR. DENTON A. COOLEY, Houston: Dr. Lam apparently has discarded esophageal bougienage in treating achalasia of the esophagus. I agree that repeated gentle dilatations with the Hurst mercury-weighted bougie was a nuisance to the patient and for the most part unsatisfactory. On the other hand, the results with the hydrostatic dilator (Plummer, Negus) have been much more encouraging, and recent reports from the Mayo Clinic and from clinics in England substantiate this point. I believe the technique of hydrostatic bougienage continues to have a place in treatment of the debilitated patient. In patients with an enormously dilated esophagus, however, one should perhaps use the cardiomyotomy of Heller or in some cases esophageal resection. The long-term results with Heller's operation are definitely better than those with cardioplasty because of the absence of acid-pepsin reflux, but I also have seen reflux esophagitis in patients after cardiomyotomy. In my opinion selection of treatment in achalasia of the esophagus should depend upon the clinical findings in the individual patient, and all patients should not necessarily be treated alike.

Chest Surveys

A Joint Committee on Diseases of the Chest, established by the American College of Chest Physicians and the American College of Radiology, has adopted a report suggesting guiding principles on the problems involved in routine chest roentgenograms in hospitals and mass chest surveys. This pooling of ideas is expected to be a continuing process, and an exchange of opinion is invited by Dr. Leo G. Rigler, Minneapolis, and Dr. Otto L. Bettag, Chicago, co-chairmen.

The current report, which has the approval of the board of regents and the board of chancellors respectively of the two organizations, recommends that each physician be encouraged to have chest roentgenograms of all of his patients; that every patient admitted to a hospital have a routine chest examination; and that follow-up for each suspected lesion seen in chest surveys should assure the patient's coming under medical supervision.

The report points out that routine chest roentgenograms are useful in screening normal from abnormal chests, but that *even 14 by 17 inch films are diagnostic aids only and should not take the place of complete clinical examinations.* Insofar as financial resources and qualified personnel permit double reading of chest roentgenograms, this procedure will increase the percentage of positive diagnoses, but a check mechanism should be instituted to eliminate the false positives. It is noted that the number of lesions overlooked in screening examinations because of clothing is so small as to make it feasible to conduct such examinations with the patient fully clothed.

The importance of continuous monitoring of all personnel associated with photofluorographic equipment to determine radiation exposure and prompt measures to correct faulty techniques or imperfect equipment permitting excessive exposure is stressed.

The committee agreed that interpretation and reporting of roentgenographic findings is a medical matter and should be handled by a responsible physician compensated like any other physician practicing his profession.

Contributions to the Memorial Library of the Texas Medical Association pay appropriate tribute upon the death of a person close to the medical profession. A card of acknowledgment and a memorial book plate help to perpetuate the memory of the deceased. Further information may be obtained from the Library, 1801 Lamar Boulevard, Austin.

SURGICAL TREATMENT OF MITRAL AND AORTIC STENOSIS

Results of 115 Valvotomies

DENTON A. COOLEY, M.D.; MICHAEL E. DeBAKEY, M.D.; RAY H. SKAGGS, M.D.; and DON W. CHAPMAN, M.D., Houston, Texas

DURING the past five years remarkable advance has been made in developing surgical procedures for the relief of congenital and acquired stenosis of the mitral and aortic valves.

TECHNIQUES

Mitral Stenosis

At the beginning of this century Brunton[4] and Samways[16] proposed that mitral stenosis, being a mechanical obstruction to blood flow, should be amenable to surgical correction, and in 1923 Cutler and Levine[12] reported the first clinical attempts at mitral valvotomy. During the ensuing years these and a few others (Allen and Graham, Pribram, and Souttar[11]) operated upon 10 patients with mitral stenosis, 2 of whom survived the operation and only 1 of whom was improved. The latter patient was operated upon by Souttar[18] to whom credit belongs for performing the first successful commissurotomy. This single case did not justify the prohibitive operative mortality of 80 per cent in the entire group, and further clinical trials were not soon forthcoming.

Recently with the inspiring leadership of Smithy,[17] Bailey,[1] Harken,[14] and Brock,[3] the surgical relief of mitral stenosis has become feasible and is now accompanied by a relatively low operative risk. The improved results have been obtained by controlling the degree of mitral regurgitation occurring after the stenosis is overcome by valvotomy. In most of the earlier attempts valvotomy was done by introducing a valvulotome into the heart in order to punch out more or less at random a piece of the stenotic valve. In the few instances where this aim was accomplished, a severe and usually fatal degree of mitral insufficiency was the consequence. With better understanding of the mechanism of valve action, a technique of valvotomy by incision at the point of fusion (commissures) of the leaflets has been developed. Thus by preserving the integrity of the leaflets and restoring valvar action, the crippling effects of mitral stenosis are overcome without the production of mitral insufficiency.

From the Departments of Surgery and Medicine, Baylor University College of Medicine, and the Jefferson Davis, Methodist, and Veterans Administration Hospitals.

This study was aided by a grant from the Houston District Chapter, Texas Heart Association.

Patients in our series were selected for mitral valvotomy if there was evidence of mitral stenosis in relatively pure form, although moderate degrees of mitral insufficiency did not preclude operation. Details of clinical evaluation of patients before operation are reported elsewhere.[6, 10] Cardiac catheterization was performed in the first 30 consecutive patients in this series,[5] but in the last 80 of the 110 patients treated by mitral valvotomy it has been done only in doubtful cases. These studies showed elevation of pulmonary arterial and pulmonary capillary pressures at rest and particularly with exercise. In most cases cardiac output during rest and exercise was below normal and in general reflected the degree of functional incapacity. As determined by the formula of Gorlin and Gorlin,[13] the cross-sectional area of the normal mitral valve is 4 to 6 sq. cm., but in our patients the calculated valve area was less than 1.5 sq. cm. Moreover, there was a relatively close correlation between the calculated valve size and the size estimated by palpation at operation.

Radiographic studies of patients for commissurotomy have been of definite value. So-called mitralization of the cardiac contour includes right ventricular and left atrial enlargement and dilatation of pulmonary arteries (fig. 1). In the absence of significant associated valvar lesions, there is no enlargement of the left ventricle. Characteristically the left atrium in mitral stenosis presents as a chamber of moderate diameter which indents the esophagus over a relatively short distance in contrast to the tremendous saclike dilatation of the left atrium which may accompany mitral insufficiency. On the other hand 2 of our patients with pronounced stenosis and mild insufficiency of the mitral valve had extreme left atrial enlargement, and both patients obtained satisfactory functional results following valvotomy. In our opinion the most reliable single diagnostic feature in selection of patients for operation is the presence of the typical rumbling diastolic murmur of mitral stenosis.

Patients were prepared for operation by digitalization, salt restricted diets, and mercurial diuretics. One hour before operation, quinidine sulfate (3 to 6 grains) was administered orally. For the most part cardiac arrhythmias other than extrasystoles were not serious

during operation, and only occasionally was quinidine lactate given parenterally or intravenously during anesthesia. Intravenous or topical administration of procaine hydrochloride and procaine amide was not employed in this series of cases.

The operative technique used in these patients has been described in detail elsewhere[9]; therefore only a brief description will be given here. The patient lies supine on the operating table with the left shoulder slightly elevated and the left arm suspended from an overhead frame. A curved anterior submammary incision is made on the left, and the chest is entered through the fourth intercostal space after the fourth costal cartilage is divided. After insertion of a self finger is inserted into the cardiac chamber.

Exploration of the atrium reveals whether or not mural thrombi are present, the elasticity and suppleness of the leaflets, the presence of calcification of the valve, and the degree of mitral regurgitation. Digital pressure and finger dissection at the commissures usually will produce the desired split, but occasionally a special valvotome may be employed to commence the valvar incision. The finger is introduced into the mitral orifice for a period not exceeding four or five heart beats in order not to impede blood flow for a significant period. After commissurotomy is accomplished, the finger is withdrawn into the atrium where a regurgitant jet of blood may be palpable during systole. Usually a carefully performed valvotomy produces no mitral insufficiency. As the finger is withdrawn from the heart, the auricular clamp is reapplied simultaneously with little or no blood loss. The auricular appendage is then amputated close to its base after the purse-string suture has been removed. The auricular opening is sutured with a double row of interrupted silk sutures, and pressures are again measured in the pulmonary artery, left atrium, and aorta for comparison with the prevalvotomy determinations. The pericardium is sutured loosely to allow drainage of pericardial fluid into the pleural cavity. An intercostal catheter is inserted into the pleural cavity for underwater drainage, and the incision is closed in layers. Ordinarily the amount of blood lost does not necessitate a blood transfusion.

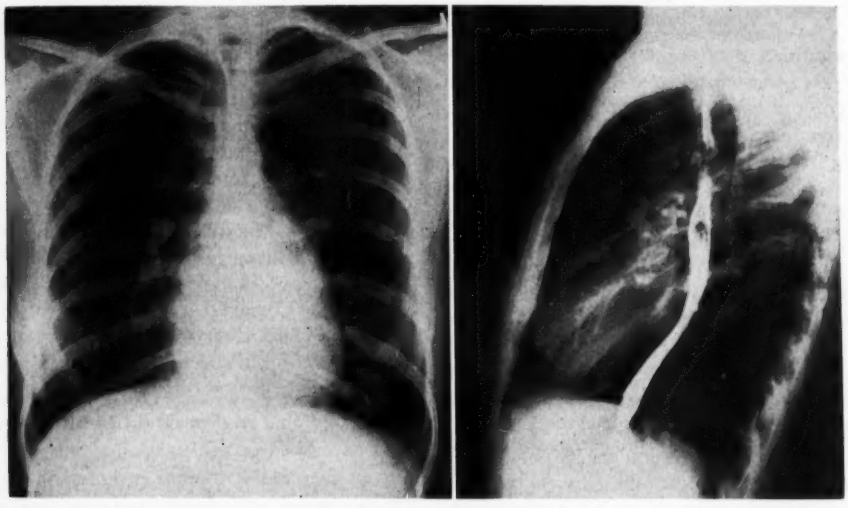

FIG. 1. Roentgenograms of the chest of a 26 year old housewife (functional class 3) demonstrating the typical mitralized contour of the cardiac silhouette. In the posterior-anterior view (*left*) the enlarged pulmonary arterial segment and left auricular appendage produce the prominence of the left cardiac border. In the lateral projection (*right*) indentation of the esophagus posteriorly by the distended left auricle is well demonstrated. The right ventricle is moderately hypertrophied, and the left ventricle is of relatively normal size. Mitral valvotomy in this case produced an excellent result.

retaining retractor, the left lung is partially collapsed, and the pericardium is entered anterior and parallel to the phrenic nerve and vessels. The incision in the pericardium provides adequate exposure of the pulmonary artery and left auricular appendage. Direct pressure determinations are made in the pulmonary artery, left atrium, and aorta. A purse-string suture of 0 black silk is placed about the base of the atrium and is used to control hemorrhage if necessary. A noncrushing clamp is placed across the base of the appendage, and near its apex an incision is made sufficiently large to accommodate snugly one's index finger. In patients suspected of having atrial thrombosis, the auricular clamp is released briefly to allow a gush of blood to wash out any loosely attached thrombi. Then as the auricular clamp is removed slowly, the index

After operation digitalization and salt restriction are maintained. Hemorrhagic shock rarely occurs. In all

patients with a sinus rhythm before operation quinidine sulfate (3 grains every four hours) is administered postoperatively to prevent the development of auricular fibrillation and is maintained for a period of four to six weeks. In 18 patients with sinus rhythm before operation fibrillation has occurred postoperatively, and in all but 1 sinus rhythm was restored by the oral administration of quinidine in larger doses. In our opinion it is desirable to maintain a sinus rhythm whenever possible. In some patients with auricular fibrillation before operation an attempt is made to convert the rhythm to sinus after operation, but conversion of the rhythm is not necessary to produce adequate functional improvement. Anticoagulants may be used in conjunction with quinidine in such cases. The usual period of postoperative hospitalization is eight days.

Aortic Stenosis

Aortic stenosis presents a more complex problem to the cardiologist and surgeon because of physiologic and pathologic features peculiar to lesions of the aortic valve. Patients may remain surprisingly well with a loud aortic murmur and marked left ventricular hypertrophy until decompensation occurs, but once this takes place there ensues an almost intractable condition which is extremely difficult to combat. The impaired function of the aortic valve interferes with coronary circulation and largely accounts for the inability of the failing left ventricle to recover its function. Thus patients should be operated upon for this condition before they have reached this precarious phase of the disease.

Aortic stenosis may be either of congenital or acquired origin. In congenital aortic stenosis the obstruction either may be in the valve itself or may occur in the subaortic zone as a fibromuscular membrane. If the stenosis is extreme, the patient may be subject to fainting attacks and even sudden death, but in most instances the congenital lesion is well tolerated. Acquired lesions of the aortic valve producing stenosis are usually the result of rheumatic fever, but there may be superimposed atherosclerosis and calcific changes particularly in the fifth and sixth decades of life. Under these circumstances the valve ring becomes rigid and immobile. Valvotomy after such changes have occurred may produce only slight benefit to the patient.

In 1913 Tuffier[26] made the first successful attempt at operative correction of aortic stenosis by digitally invaginating the aortic wall and dilating the valve. Interest in the surgical approach to this problem was revived by the late Horace Smithy,[24, 25] who himself died in 1948 of aortic stenosis. Numerous techniques for aortic valvotomy have been proposed.[19-23] The most satisfactory method in our opinion is described by Bailey, Redondo Ramirez, and Larzelere,[19] in which procedure a special valvotome is passed through the wall of the left ventricle into the stenotic valve. The head of the valvotome is composed of three expanding parts which engage the fused commissures and when opened split the cusps apart. Although a moderate degree of aortic insufficiency may be produced if the valve leaflets are ruptured or torn, relief of aortic stenosis may be obtained with associated clinical improvement if the valve is sufficiently supple to allow it to open and close after valvotomy is accomplished.

CLINICAL RESULTS

This report deals with 110 patients undergoing mitral valvotomy and 5 patients undergoing aortic valvotomy between March, 1951, and November, 1953. Although aortic and mitral stenosis occurred in combination in several instances, simultaneous aortic and mitral valvotomy was attempted only once.

Mitral Stenosis

In the series of 110 patients operated upon for mitral stenosis, there have been 10 deaths occurring at varying intervals following operation. In the 100 survivors an attempt has been made to estimate the degree of improvement, based on relief of symptoms, functional improvement, and when possible, information obtained by cardiac catheterization. Results were considered good in 74 of the survivors, fair in 19 patients, and poor in 7. In the group of patients whose results were classified good, there were many with excellent and striking restoration of functional activity, and in these, cardiac murmurs were almost eliminated. In others, the functional improvement was exceptionally good, but there was less striking alteration of the cardiac murmurs.

Deaths.—The 10 patients who died in this series of 110 mitral valvotomies were severely incapacitated, class 4 patients, most of whom were in the advanced age group (table 1). Moreover, all of these patients had had fibrillation before operation and 2 had previously had peripheral embolism. Of prime importance in the fatal cases was the fact that thrombosis and embolism was the probable cause of death in 9 patients. Thrombosis of a calcified mitral leaflet was evident in 2 patients and produced progressive valvar occlusion. Peripheral embolism definitely contributed to the death of 3 patients, and cerebral embolism was presumed to have been present in 2 others. Massive pulmonary embolism caused death in 2 patients, thirty hours and six days respectively after operation, and was the presumptive cause of death in a third patient who died suddenly at home one month after

operation. One patient who before operation had severe mitral insufficiency and almost intractable decompensation died of pulmonary edema four days after exploration of the mitral valve.

Severity of Cardiac Condition.—Functional rating of patients is similar to the classification proposed by the New York Heart Association.

As shown in table 2, 40 per cent of the patients in this series were in class 4, and 93 per cent were in either class 3 or 4. Most of the patients were severely disabled, and many had been totally bedfast from three to eighteen months. The percentage of good to fair results was higher in class 3 than it was in class 4, although good results were obtained in about 60 per cent of the patients in class 4. This high percentage of good results in class 4 patients would appear to offset the fact that all deaths occurred in this group. Operation thus seems to be justified in these patients who are otherwise destined to chronic invalidism or premature death.

Age.—The ages of the patients varied over a wide range between 16 years and 58 years, with 69 patients being less than 40 years of age and 41 patients more than 40 years old.

In all patients less than 25 years of age the possibility of reactivation of the rheumatic process by operation deserves consideration. All of our younger patients therefore were observed carefully for varying periods before operation to detect symptoms of rheumatic activity. Four patients in the younger group following operation have had transient symptoms suggestive of a recrudescence of rheumatic fever. In general, however, the most satisfactory results were obtained in patients between 25 and 35 years. In patients more than 40 years of age good results were obtained in 22 (more than half), and there were 7 deaths. In patients less than 40 years of age good results were obtained in 57 (about four-fifths) and 3 patients died. Although the fatality rate in the older patients was higher than in the younger group, the percentage of good results in the patients above 40 years justifies operation. The oldest patient in this series was 58 years old, and an excellent result was obtained. Three other patients beyond 50 years of age have obtained good symptomatic relief from operation.

Previous Embolism.—Peripheral embolism occurred before operation in 16 patients, and in 7 of these there were multiple emboli. Ten patients had cerebral emboli producing neurologic sequelae such as hemiparesis and partial aphasia. In others the emboli lodged in the vessels of the extremities or viscera (spleen, kidney, intestine). Seven patients were suspected of having pulmonary infarctions before operation, but in 4 of these the evidence was equivocal. Twelve of the patients with previous peripheral emboli had fibrillation at the time of operation. In this series of 16 patients with peripheral emboli prior to operation, 3 showed evidence of embolization after operation, 2 of which were fatal. Thus among the 14 patients who survived operation only 1 subsequently developed this complication. It appears, therefore, that peripheral embolism in the presence of mitral stenosis is actually an indication for valvotomy and offers best protection against future embolization.

TABLE 1.—*Analysis of 10 Deaths in Series of 110 Mitral Valvotomies.*

Case	Age (Yr.)	Sex	Functional Classification	Auricular Fibrillation	Operative Findings	Survival	Cause of Death
1	53	M	4	+	Mural thrombi; calcified valve	30 hr.	Aortic "saddle" embolus
2	51	M	4	+	Calcified valve	4 mo.	Thrombosis mitral valve, aortic "saddle" embolus
3	49	F	4	+	Mural thrombi; calcified valve	3 days	Cerebral embolus
4	31	F	4	+	Marked mitral insufficiency	4 days	Congestive failure
5	47	F	4	+ (Flutter)	Calcified valve	1 mo.	Possible pulmonary embolus
6	33	M	4	+	Mural thrombi; temporary carotid occlusion	16 hr.	Cerebral anoxia; cerebral embolus(?)
7	47	F	4	+	Mural thrombi; calcified valve	10 days	Cerebral infarct; pulmonary congestion
8	37	M	4	+	Mural thrombi; calcified valve	6 days	Massive pulmonary embolus
9	44	M	4	+	Mural thrombi; calcified valve	30 hr.	Massive pulmonary embolus
10	41	F	4	+ (Intermittent)	Marked calcification	10 days	Thrombosis mitral valve

TABLE 2.—*Functional Classification (New York Heart Association) in 110 Patients Undergoing Mitral Valvotomy.*

Class	No. Cases	%
1	0	0
2	8	7
3	58	53
4	44	40
Total	110	100

Auricular Fibrillation.—Before operation 54 patients had auricular fibrillation, and 56 patients had sinus rhythm. Good results were obtained in 32 patients with auricular fibrillation and in 42 patients with sinus rhythm. Fibrillation was present in all patients who died. These results indicate that although auricular fibrillation is not a contraindication to valvotomy, it apparently does increase the operative risk and slightly diminishes the possibility of obtaining a

good functional result. On the other hand, the proportion of good results in the patients with fibrillation is sufficiently high to justify operation.

Of real importance in auricular fibrillation is the frequency of the presence of mural thrombosis in the auricle. In the 54 patients with fibrillation, mural thrombi were demonstrated in the left atrium or auricular appendage in 17 instances. In 4 of these embolism was discovered soon after operation. In 1 the embolus involved only pulmonary vessels, indicating that there may have been right atrial thrombosis as well as left. Measures taken at operation to reduce the danger of embolism have been employed in all patients with auricular fibrillation and mural thrombosis. In most cases the valve was approached through the left auricular appendage whether or not it was suspected of containing thrombi. Access to the valve was gained through the superior pulmonary vein in 2 cases where the appendage was contracted and fibrosed or calcified, preventing its use as a means of safe entry into the heart.

In 6 patients the left atrium was completely lined by thrombi in varying stages of organization, but valvotomy was performed in all. Death from cerebral embolism occurred in 1 of these patients, and the others recovered uneventfully. Although it seems desirable during intracardiac manipulation to have the anesthetist compress the carotid arteries to prevent cerebral embolization, this technique failed to prevent fatal embolism in 1 case. The technique suggested by Bailey[2] for temporary complete occlusion of the carotid circulation within the thorax has been employed in 9 patients but also may be unreliable and even dangerous. If intrathoracic occlusion of the carotid arteries is to be used under these circumstances, the period of occlusion should be less than two minutes and full oxygenation of the circulating blood must be assured before the vessels are clamped. Obviously, temporary carotid occlusion does not preclude the possibility of emboli lodging elsewhere in the arterial system. Anticoagulants have been employed after operation in some of the patients but not in a sufficient number to permit definite conclusions.

Valvar Calcification.—The more extensively calcified mitral valve may be demonstrated by fluoroscopy and roentgenography prior to operation, but from our experience this finding does not contraindicate exploration. Calcification of the mitral valve was discovered at operation in 53 patients (48 per cent) in this series. In some instances calcification was extensive and involved the valve leaflets as well as the commissures. In a few, exuberant calcification was palpated at the commissures, usually the anterolateral one, but surprisingly good results have been obtained in these patients. When a split is made through a calcified commissure, mild but not incapacitating mitral insufficiency may be produced. In no instance did embolization of a calcified fragment occur or did bacterial endocarditis supervene. Of the 53 patients in whom calcification of the valve was discovered at operation, good results were obtained in 31 patients, fair results in 10 patients, poor results in 4, and 8 patients died.

Associated Valvar Lesions.—Although a moderate to marked degree of mitral insufficiency was present in about 25 patients operated upon, concomitant lesions of other valves were infrequent. Three patients had a moderate degree of associated aortic stenosis, but in none was simultaneous mitral and aortic valvotomy done. One of these patients died ten days after operation of thrombosis of the valve and sudden occlusion of the mitral orifice. The other 2 patients obtained excellent functional results in spite of the persistent aortic stenosis, although subsequent aortic valvotomy may be required in 1 of these patients. Aortic insufficiency was not demonstrated in any patient. Increasing experience indicates that a Graham-Steele murmur of functional pulmonic insufficiency is a frequent finding in pure mitral stenosis (26 cases in our series) and mimics the murmur of aortic insufficiency. When pulmonary arterial pressure is reduced after commissurotomy, the Graham-Steele murmur disappears. Before operation none of the patients was suspected of having associated tricuspid or pulmonic valvar lesions.

In general, the finding of a grade 1 or 2 mitral systolic murmur with mild left ventricular enlargement does not preclude surgical treatment in patients in whom mitral stenosis is the predominant lesion. A carefully performed mitral valvotomy seldom increases the degree of mitral insufficiency. In 5 patients, however, it was thought that mitral regurgitation was increased by the valvotomy, but in these instances the relief of mitral stenosis produced gratifying functional improvement. Commissural dissection should be performed with extreme care so that valvar action is restored and the stenosis relieved without producing an incompetent valve. Mild mitral insufficiency frequently accompanies mitral stenosis, but even in these cases valvotomy may produce striking functional improvement.

Pregnancy and Mitral Stenosis.—Five patients in this series were pregnant at the time of valvotomy. The first was a 28 year old primigravida in the fourth month of pregnancy who was experiencing progressive disability and mild decompensation. Valvotomy produced an excellent functional result, and five months later she delivered uneventfully a 7 pound normal girl.[8] A second patient was a 24 year old primigravida in the third trimester of pregnancy. She

had experienced 2 attacks of severe pulmonary edema in spite of a rigid regimen to control cardiac symptoms. Operation was performed during the thirty-sixth week of pregnancy, and sixty-two hours later she delivered a 5 pound 10 ounce boy. Mother and son were discharged from the hospital five days after delivery in good condition. Three other patients have undergone valvotomy during pregnancy, and all subsequently have withstood delivery uneventfully.

This experience indicates that mitral valvotomy is not only feasible during pregnancy but should be attempted in those instances of class 3 and 4 cardiac patients if the anticipated risk of decompensation during delivery appears to be serious. If commissurotomy is to be done under such circumstances, the optimum time for operation is before the fifth month of pregnancy. Further experience is necessary before other specific recommendations can be made in regard to cardiac surgery during pregnancy.

During operation the pressures in the left auricle, pulmonary artery, and a systemic artery have been measured before and after valvotomy. An immediate fall in mean pulmonary artery pressure and also in the left atrial pressure of 15 or 20 mm. of mercury represents a good immediate response. In several patients whose mean pulmonary arterial pressure was exceedingly high (80 to 100 mm. of mercury), the pressure did not fall immediately after commissurotomy in spite of a significant drop in left atrial pressure. This indicates that in some patients with mitral stenosis the vascular changes in the pulmonary vessels may delay the restoration of normal pulmonary pressure after operation. These vascular changes have been described in detail previously by Parker and Weiss.[15] In most of our patients such histologic changes have been investigated by lung biopsy and will form the basis of a subsequent report.

Recurrence of mitral stenosis after valvotomy has not taken place in any of our patients and must be rare, probably because the rheumatic infection is quiescent in patients selected for operation.[7] Other factors, too, undoubtedly are responsible for this gratifying observation. Histologic studies of the amputated auricular appendages reveal endocardial thickening and subendocardial infiltration with round cells in most specimens, but Aschoff nodules were encountered in only a few specimens, and other histologic findings of acute rheumatic carditis have not been demonstrated.

Aortic Stenosis

In 5 patients aortic valvotomy has been performed by the transventricular route using the Donaldson-Bailey valvotome. Three patients have survived operation and are improved, and 2 have died (table 3). In the first patient, a 40 year old taxi driver, the aortic stenosis was considered to be postrheumatic and was markedly incapacitating. Direct radial pulse tracings made before and after valvotomy indicated considerable improvement in cardiac output as evidenced by the change in the contour of the systolic component. There also was a general increase in functional capacity following operation, and no aortic insufficiency was produced.

TABLE 3.—*Analysis of 5 Cases of Aortic Valvotomies.*

Case	Age (Yr.)	Sex	Etiology	Operation	Remarks
11	40	M	Rheumatic	2-6-53	Uneventful recovery; improved
12	6	M	Congenital	2-17-53	Uneventful recovery; improved
13	27	F	Rheumatic	3-10-53; combined aortic and mitral valvotomy	Died; cardiac arrest and fibrillation during chest incision
14	10	M	Congenital	3-19-53	Recovered and improved; moderate aortic insufficiency
15	46	F	Rheumatic; calcific	6-12-53	Died; ventricular fibrillation during insertion of valvulotome

The 2 deaths occurred during operation. The first of these was a 27 year old woman who had severe cardiac dysfunction (class 4) with combined aortic and mitral stenosis. For the previous two months she had been in moderate to severe intractable congestive failure and required almost continuous oxygen therapy. Operation was undertaken as a last resort. Cardiac arrest occurred during induction of anesthesia. Hasty aortic and mitral valvotomies were done, and both valves were found to be markedly stenosed. Subsequently feeble heart action was restored temporarily by cardiac massage, but effective cardiac action was not maintained, and the patient died. The other seriously ill patient, who was 46 years old and had aortic stenosis and congestive failure, developed ventricular fibrillation during insertion of the valvotome. Valvotomy with subsequent attempts at cardiac resuscitation was unsuccessful.

In patients with pronounced left ventricular hypertrophy, the myocardium appears to be very susceptible to ventricular fibrillation possibly because of the relative coronary insufficiency and myocardial ischemia. The manipulation required to insert the valvotome in such instances is likely to produce fibrillation in spite of the use of depressants such as quinidine or procaine amide. Thus it appears that valvotomy should be recommended to patients with acquired aortic stenosis before advanced stages of deterioration and myocardial decompensation occur. From our study of autopsy specimens it appears that

operation upon patients with advanced calcific stenosis is unsatisfactory. In 1 such specimen the valvulotome was badly damaged by attempting to fracture a densely calcified valve and withdrawal of the instrument through the ventricular wall would have injured seriously the myocardium, possibly to a fatal degree. After valvotomy upon a calcified, rigid valve, there is a tendency for it to spring back into its original shape without relief of stenosis.

Two patients with congenital aortic stenosis have been operated upon with gratifying results. The first patient was a 6 year old boy who had several episodes of syncope prior to operation. Valvotomy was well tolerated, and since surgery he has remained entirely well. The second patient with a congenital stenosis was a 10 year old boy with moderate cardiac enlargement and physical incapacity. Temporary cardiac arrest occurred during valvotomy, but after the instrument was withdrawn, cardiac massage restored heartbeat and recovery was uneventful. Since operation this patient has developed mild aortic insufficiency, but he is active and free of symptoms.

In patients with congenital aortic stenosis the obstruction may be located in the valve itself or in a subvalvar zone where a fibromuscular membrane may obstruct the aortic outflow tract. In the strictly valvar lesion aortic insufficiency of moderate degree may occur after valvotomy whereas in subaortic stenosis the valve ring may be guarded by a relatively normal valve. Rupture of the subvalvar membrane in this instance would not produce aortic regurgitation. Unfortunately, methods to make this distinction between valvar and subvalvar stenosis prior to operation have not been satisfactory, and it appears to be justifiable to perform a surgical dilatation of the valve ring in those patients with progressive severe symptoms of congenital aortic stenosis even though mild aortic regurgitation may be produced. Further experience with congenital aortic stenoses is necessary before specific recommendations may be given in the selection of such cases for operation.

SUMMARY

During the past five years surgical procedures have been developed for the relief of congenital and acquired stenosis of the mitral and aortic valves.

This report deals with 115 patients undergoing either mitral or aortic valvotomy between March, 1951, and November, 1953. In 110 valvotomies for mitral stenosis there have been 10 deaths giving a fatality rate of 9 per cent. Eight patients died during the first ten days after operation, and death occurred in 2 patients one and four months respectively following operation. Thrombosis and embolism accounted for death in 9 of the 10 patients who died following operation. Clinical results in 74 per cent of the survivors was good to excellent, and many were restored to normal activity with nearly complete disappearance of murmurs.

Although the risk of operation is somewhat increased in severely incapacitated patients with mitral stenosis, particularly in the advanced age group, valvotomy has produced striking improvement in the majority of such patients. Ages of patients ranged between 16 and 58 years, but good results were obtained more uniformly in patients 25 to 35 years of age. Other factors such as history of previous embolism, auricular fibrillation, valvar calcification, and associated valvar lesions deserve consideration in the evaluation of patients for valvotomy but should not contraindicate operation in the presence of mitral stenosis. Five pregnant patients with mitral stenosis have been operated upon successfully with subsequent normal delivery. The tendency for severe stenosis to recur after successful mitral valvotomy appears to be relatively insignificant.

Aortic stenosis is a more complex problem to the cardiologist and surgeon, but valvotomy may relieve symptoms of reduced cardiac output in properly selected cases. In 5 patients undergoing aortic valvotomy, the lesion was acquired in 3 and congenital in 2. Two critically ill patients with acquired stenosis did not survive operation, but I survived and was improved. Valvotomy produced gratifying relief of symptoms in 2 patients with congenital aortic stenosis. Patients with aortic stenosis should be considered for operation before the development of marked cardiac enlargement and accompanying coronary insufficiency.

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HEART DISEASE IN PREGNANCY

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HEART disease in pregnancy has assumed a more important position as the frequency of other causes of maternal mortality has been reduced. As a result, cardiovascular disease is responsible for about one-fourth of maternal deaths occurring in this country today. There are signs which suggest that this mortality is not irreducible.

The fundamental studies regarding the basic physiologic changes in the cardiovascular system during pregnancy are becoming more widely appreciated. This knowledge, together with the growing awareness of the need for cooperative, careful prenatal attention by the obstetrician and internist or cardiologist, has already brought the mortality down to such low figures as the 0.64 per cent recently reported by Gorenberg and Chesley.[9] Furthermore, recent advances in the treatment of mitral stenosis, coarctation of the aorta, patent ductus arteriosus, tetralogy of Fallot, and subacute bacterial endocarditis tend to make pregnancy a lesser hazard in certain patients, although it is true that new and often trying problems confront the physicians who attend these patients. For many years rheumatic heart disease has been responsible for from 90 to 95 per cent of the total of cardiac patients who become pregnant, with congenital heart disease accounting for 2 or 3 per cent and miscellaneous disorders constituting the remainder. These proportions undoubtedly will change as surgical management prolongs the life of patients with congenital cardiac defects and preventive measures of various types reduce the incidence of rheumatic heart disease.

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CARDIOVASCULAR LOAD OF PREGNANCY

The burden which pregnancy imposes upon the heart may be understood from table 1, which summarizes the changes occurring in the cardiovascular system. Although the causes of these alterations are not fully understood, they are probably related to endocrinologic modifications attending the pregnancy with accompanying retention of salt and water. The increased blood volume is primarily an increase in plasma volume. Furthermore, the studies recently reported by Berlin and his associates[3] using erythrocytes labeled with radioactive phosphorus (P^{32}) have shown a definite decrease in total red cell volume in the first trimester followed by a slow rise to a maximum in the ninth month. This is followed by a decrease prior to delivery and a further 300 cc. fall at delivery. The plasma volume begins to rise in the second month and also reaches a maximum at the ninth month. Again, a fall precedes delivery with an additional 700 cc. drop at delivery. Berlin and his coworkers reported an average increase of more than 1,300 cc. in the total blood volume during pregnancy.

TABLE 1.—*Changes in Circulatory Dynamics in Pregnancy.*

Increased total blood volume (1-1.3 L.)
Increased cardiac output (25-40%)
Increased cardiac work (50%)
Increased oxygen consumption (15-25%)
Accelerated circulation rate
Decreased arteriovenous oxygen difference

Burwell and his associates[6] developed the hypothesis that the placenta behaves much like an arteriovenous aneurysm and that the circulatory dynamics are altered accordingly. It is believed that this alteration may be one of the participating mechanisms, but the retention of salt and water is as important, or more so. This latter circumstance lends itself to alteration by medical means if the patient should develop congestive heart failure during the pregnancy.

The feature which seems of greatest practical importance in the cardiovascular changes accompanying pregnancy is that the load upon the heart reaches a peak at from thirty-two to thirty-six weeks. There is then a gradual drop in the last lunar month. A curve

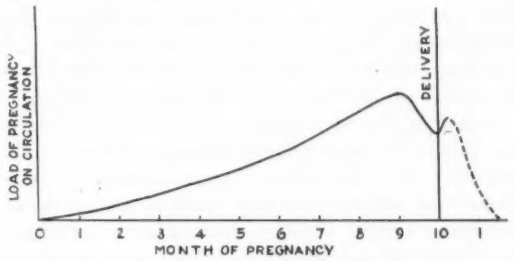

FIG. 1. A graph representing the postulated load of pregnancy upon the circulation. Note that the peak occurs at the ninth lunar month and a significant fall precedes delivery. A small second peak occurs in the early postpartum period. (Modified from Hamilton, B. E., and Thomson, K. J.: Heart in Pregnancy and Childbearing Age, Boston, Little, Brown and Co., 1941.)

representing this load has been developed by Hamilton and Thomson[10] (fig. 1). If this curve is kept in mind, it is of help in managing pregnant cardiac patients. For many years when congestive heart failure developed during pregnancy, there was an inclination to terminate the pregnancy, often by cesarean section, at exactly the point on this curve when the load is greatest to spare the patient what was thought to be the greater burden at full term. It is appreciated now that the load lessens markedly during the last month and that the patient is really in a more favorable state at full term.

When the load of pregnancy upon the circulation is understood, it becomes clear why many normal women develop certain manifestations which suggest heart disease. These include dyspnea, palpitation, pulmonic and apical systolic murmurs, apical third heart sounds, tachycardia, full pulses, and a seeming increase in the size of the heart. When patients have not been examined prior to pregnancy, it is necessary to evaluate these manifestations with some care in order to avoid an erroneous diagnosis of structural heart disease.

EVALUATION OF PATIENT

Several plans for evaluating particular patients in pregnancy have been proposed. Such systems of classification are of great usefulness since they permit the attending physicians to anticipate difficulties, particularly congestive heart failure, in certain classes of patients. In addition they are of help in advising patients, whether or not the burden of pregnancy should be undertaken. The New York Heart Association developed a classification applicable to patients with heart disease.[15] There are four classes of patients under this system according to their functional capacity. Class 1 consists of those persons with heart disease who have no limitation of physical activity. Class 2 is made up of those who have slight limitation of physical activity. Class 3 is for those patients who have marked limitation of physical activity, and class 4 consists of those patients who are unable to carry on any physical activity without discomfort. General experience has shown that patients in classes 1 and 2 usually tolerate pregnancy well. The risk of pregnancy, particularly the development of congestive heart failure, is much greater for patients in classes 3 and 4.

TABLE 2.—*Duration of Rheumatic State and Incidence of Heart Failure During Pregnancy.*

Duration of Rheumatic State (Yr.)	Pregnancies (No.)	Pregnancies with Failure (No.)	(%)
0-9	40	2	5
10-19	77	15	19.3
20-29	36	14	38.8
30+	6	5	83.3

Hamilton and Thomson[10] proposed classification of cardiac patients into unfavorable and favorable groups insofar as the hazard of pregnancy is concerned. The unfavorable category is made up of those who have (1) signs or history of heart failure, (2) a dangerous disorder of the heart beat such as auricular fibrillation, or (3) a complicating serious disease. Women with heart disease who do not have any of these criteria are considered favorable risks. The experience of Hamilton and Thomson with the first 750 cardiac patients delivered at the Boston Lying-In Hospital indicated a mortality rate of 3.5 per cent for the patients in the favorable class and 16.7 per cent for those in the unfavorable class.

It is generally believed that rheumatic fever is a disease which has a rather definite natural history consisting of four phases: (1) the initial attack characterized by arthralgia, carditis, chorea, and other rheumatic manifestations; (2) a subacute phase during which recrudescences are frequent; (3) an inactive phase, usually from puberty to the fourth or fifth decade, during which functional capacity is usually good; and (4) a final phase characterized by pro-

gressive reduction in functional capacity with ultimate congestive heart failure and death. The development of auricular fibrillation often signals the onset of the fourth phase.

Bunim and Rubricius[5] have recommended that by establishing the position of the patient in the natural course of rheumatic heart disease, the possibility of congestive failure can be prognosticated and the risk of pregnancy may be estimated. The risk of pregnancy, particularly the possibility of congestive failure, is much greater when the patient has entered the fourth phase of rheumatic disease. In 1950, Correll and I[7] reported observations made in 52 women with rheumatic heart disease who had four or more pregnancies. It may be seen from table 2 that congestive failure occurred in only 5 per cent of those pregnancies which occurred within nine years of the onset of rheumatic fever, whereas failure developed in 83 per cent of the pregnancies which occurred thirty or more years after the onset of rheumatic fever.

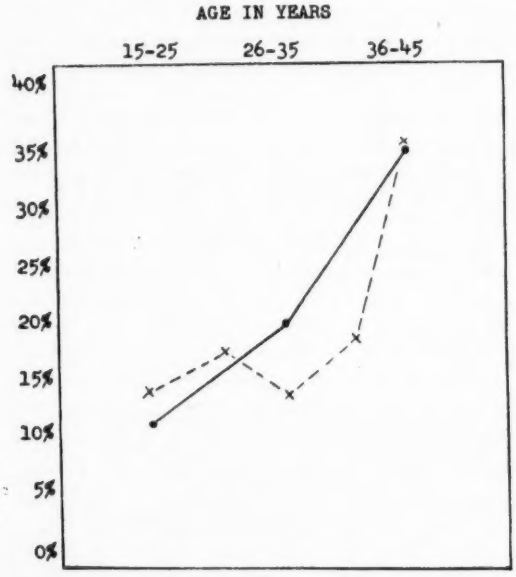

FIG. 2. A graph showing the incidence of congestive heart failure during pregnancy in patients with heart disease, which indicates the increasing frequency of this complication as the patient grows older. Solid line represents studies of Correll and Rosenbaum.[7] Dotted line indicates observation of Hamilton and Thomson.[10] (After Correll, H. L., and Rosenbaum, F. F.: Multiple Pregnancies in Patients with Rheumatic or Congenital Heart Disease, Am. Heart J. 39:283-297 [Feb.] 1950.)

It is of value to survey a single patient from many points of view when the risk of pregnancy is being estimated. In addition to classifications such as those described, specific features may be evaluated. The age of the patient is probably more important than any other single factor. Hamilton and Thomson observed a sharp increase in the incidence of heart failure in pregnancy at age 35 years, the frequency of this complication doubling in the older patients. Bunim and Rubricius[5] have confirmed this, and our observations (Correll and Rosenbaum[7]) in the special group of patients mentioned earlier support this belief (fig. 2).

A feeling in some quarters that the outlook is more serious if the size of the heart exceeds the predicted normal by 10 per cent exists; however, there is some difference of opinion on this point.[4, 5] I recently have followed the progress of a woman, aged 33 years, with aortic stenosis and insufficiency and mitral insufficiency through her third pregnancy. The heart was at least 60 per cent larger than the predicted normal and electrocardiograms showed evidence of pronounced left ventricular hypertrophy. Nevertheless, the pregnancy was uneventful and delivery uncomplicated. The first attack of rheumatic fever had occurred twenty-four years earlier. Striking exceptions to rules, such as this, are uncommon.

There is also no complete agreement concerning the prognostic significance of the type of valvular involvement.[5, 10] One of the greatest hazards is acute pulmonary edema which may occur without warning and prove rapidly fatal in women with mitral stenosis. Bramwell[4] and Jones[11] both have called attention to this condition and emphasized that it is apt to occur when normal sinus rhythm is present, probably as the result of abrupt left auricular failure.

Auricular fibrillation increases the death rate by as much as four times.[4] In one large series of cardiac patients with unfavorable prognosis, who had auricular fibrillation either established or transient, the maternal death rate was 33⅓ per cent.[10] However, Bramwell recently has expressed the opinion that auricular fibrillation is not a bar to pregnancy if observation and adequate treatment are possible. It is my own belief that this disorder of rhythm is both an unfavorable sign and an index of the final phase of the natural history of rheumatic disease. Pregnancy should be undertaken or continued in the face of this arrhythmia only with a full appreciation by all concerned of the risk involved.

There is a growing body of evidence that parity per se is not a vitally important factor in prognosis in these patients.[9, 10, 11] Our study (Correll and Rosenbaum[7]) was initiated as an attempt to determine the relationship of parity to prognosis. We observed 10 patients who had had ten or more pregnancies carried to term. No clear correlation could be worked out between the incidence of heart failure and increasing parity (table 3). It must be borne in mind that with each succeeding pregnancy the patient is older and the rheumatic state of greater duration, factors which

TABLE 3.—*Correlation of Increasing Parity with Incidence of Heart Failure.*

Para	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Total patients	53	53	53	53	39	31	27	22	13	10	4	2	1	1	1	1
No. with failure	6	5	3	6	5	4	5	3	1	2	1	0	0	0	0	0
Per cent with failure	11.3	9.4	5.7	11.3	12.8	13.0	18.5	13.6	7.7	20	25	0	0	0	0	0

After Correll, H. L., and Rosenbaum, F. F.: Multiple Pregnancies in Patients with Rheumatic or Congenital Heart Disease, Am. Heart J. 39:283-297 (Feb.) 1950.

already have been shown to affect appreciably the course of pregnancy.

If the patient has had previous pregnancies, a detailed description of their course is helpful. If heart failure has occurred in a previous pregnancy, there is a 50 to 75 per cent chance of its recurrence in succeeding pregnancies.[7, 8] In some series this recurrence rate has been as high as 89 per cent.[19] Not only does congestive failure recur but there is evidence to suggest that it appears earlier and is of more severe degree with each successive pregnancy. It is believed by many observers that patients with acute rheumatic fever present an unusually high risk, and if the diagnosis can be made unequivocally early in pregnancy, termination is indicated.[13] In evaluating any patient's status it is well to estimate the functional capacity on the basis of what the patient was able to do in her ordinary routine of living prior to the pregnancy.[4] It is well to keep in mind, however, that the functional capacity may change from time to time during the pregnancy.

MANAGEMENT OF PREGNANCY

The most important principle to keep in mind in the management of these patients is that they must be seen often and watched carefully. It is my own practice on each visit to review the symptoms, asking particularly about unusual dyspnea, cough, hemoptysis, palpitation, sudden gain in weight, or unexpected edema of the ankles. Furthermore, there should be a careful examination, including measurement of the blood pressure and vital capacity; and search for basillar rales; changes in the cardiac sounds, murmurs, or rhythm; and enlargement of the liver. The appearance of basillar rales and a gradually diminishing vital capacity may foretell congestive failure before the patient is aware of any change in symptoms. Patients with heart disease should be advised to limit weight gain to 20 pounds, to restrict the sodium intake moderately throughout pregnancy, to do that amount of housework and physical activity which can be performed with ease and comfort and without undue fatigue or dyspnea, and to take daily rest periods. If any infection occurs, the patient should take to her bed at once and appropriate treatment should be started. Respiratory infections are particularly prone to precipitate congestive heart failure in these patients.

If congestive heart failure appears, the patient should be hospitalized and treated in the usual way. It is probably wise to keep such patients in the hospital throughout the remainder of pregnancy. This is often difficult because of the economic burden imposed, and patients resist such confinement because they feel so well when the heart failure is relieved. The following case illustrates this point.

CASE 1.—L. Z., aged 34 years, was seen initially April 25, 1947, during the fifth month of her third pregnancy. The previous pregnancies eight and five years earlier were associated with considerable dyspnea during the last trimester. Features of mitral stenosis were present (fig. 3).

Hemoptysis and severe dyspnea appeared abruptly during

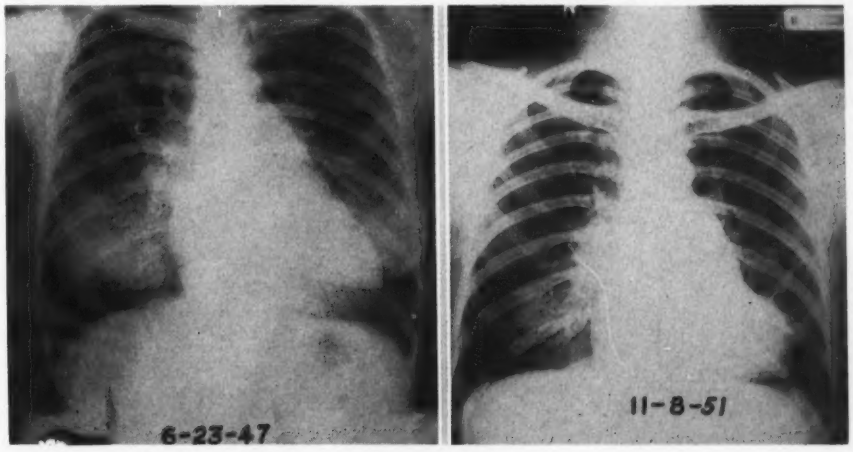

FIG. 3. Case 1. Woman, aged 34 years, with rheumatic mitral stenosis. *Left.* Roentgen study of the heart during the seventh month of the third pregnancy. The cardiac silhouette has typical mitral configuration. Delivery was complicated by nearly fatal acute pulmonary edema. *Right.* Roentgen study of the heart four months after successful mitral valvulotomy shows some decrease in cardiac size.

the seventh month, and the patient was admitted to the Milwaukee Hospital. Prompt improvement followed application of the usual measures. The patient left the hospital at her own insistence three weeks after admission. There was some increase in peripheral edema thereafter but the lungs remained clear.

Six weeks after discharge she was readmitted for induction of labor. Shortly after rupture of the membranes with subsequent rapid progress, the patient developed overwhelming acute pulmonary edema so severe that she appeared moribund. Delivery was so rapid and the patient's condition so poor that the spinal anesthesia which was planned was not used. Atropine, aminophyllin, venous tourniquets, and oxygen administered under positive pressure did not alter her condition. Improvement followed venesection with withdrawal of 500 cc. of blood.

The patient subsequently recovered and went on to develop severe, chronic congestive failure. A mitral valvulotomy was performed by Dr. Dwight Harken July 5, 1951, and the patient currently is in satisfactory condition.

If this patient had been kept in the hospital from the time of first admission until the delivery, it is probable that acute pulmonary edema would have been avoided. This case illustrates the suddenness with which the complication may arise in pure mitral stenosis and the value of phlebotomy as well. I have seen a similar case in an elderly primipara with mitral stenosis and auricular fibrillation who undertook a pregnancy despite the advice of many physicians to the contrary. Phlebotomy was again life saving. If it is remembered that the total blood volume is increased by more than one liter during pregnancy, the load upon an embarrassed circulation may be appreciated and the relief afforded by venesection understood. It is a measure which too often is not considered or felt to be outmoded.

It is perhaps well to point out that because of this increase in the total blood volume, the parenteral administration of fluids or blood must be undertaken with great caution and a full understanding of the hazard. Acute pulmonary edema of alarming degree has been observed when such measures have been used to overcome dehydration or anemia in pregnant women with mitral stenosis.[17]

It is often gratifying to observe how well some patients will tolerate the load of pregnancy when at first appraisal the risk would seem prohibitive. This point is illustrated by the following case.

CASE 2.—J. R., aged 21 years, was first seen January 3, 1949, during the third month of her first pregnancy. A murmur had been first detected when she was six months of age; a deformity of the spine had appeared at the age of 10 years. There was an extremely severe right dorsal, left lumbar kyphoscoliosis, and the cardiac findings were characteristic of an interventricular septal defect (fig. 4).

The patient was seen at two week intervals throughout pregnancy. The vital capacity, which was 1,000 cc. on first determination, never fell below 850 to 900 cc. A few, transient rales, moderate dyspnea, and fatigue appeared during the last trimester, but these changes were never severe enough to cause concern. A normal infant was delivered by mid-forceps July 20, 1949, and the postpartum period was uneventful.

In this instance the risk of continuation of the pregnancy was discussed at length with the patient and her husband. She was cooperative in following the recommendations given her by the obstetrician and me. Such cooperation by all concerned is imperative, if such high risk patients are to be carried through pregnancy successfully.

Patients with congenital heart disease present certain special problems. Girls with tetralogy of Fallot reach the childbearing age frequently, yet pregnancy can be a great burden for them. The development of surgical procedures for the amelioration of this condition has lessened this burden. Successful outcomes

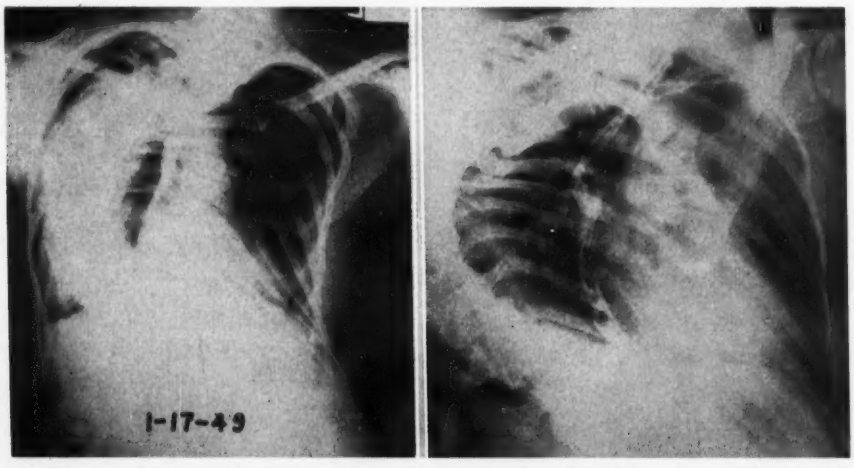

FIG. 4. Case 2. Woman, aged 21 years, with interventricular septal defect and severe kyphoscoliosis. Roentgen studies of the heart and thorax during the third month of the first pregnancy. *Left.* Antero-posterior view. *Right.* Right anterior oblique view. Gross distortion of the spine is easily seen. An uncomplicated pregnancy concluded with a normal fetus delivered at full term.

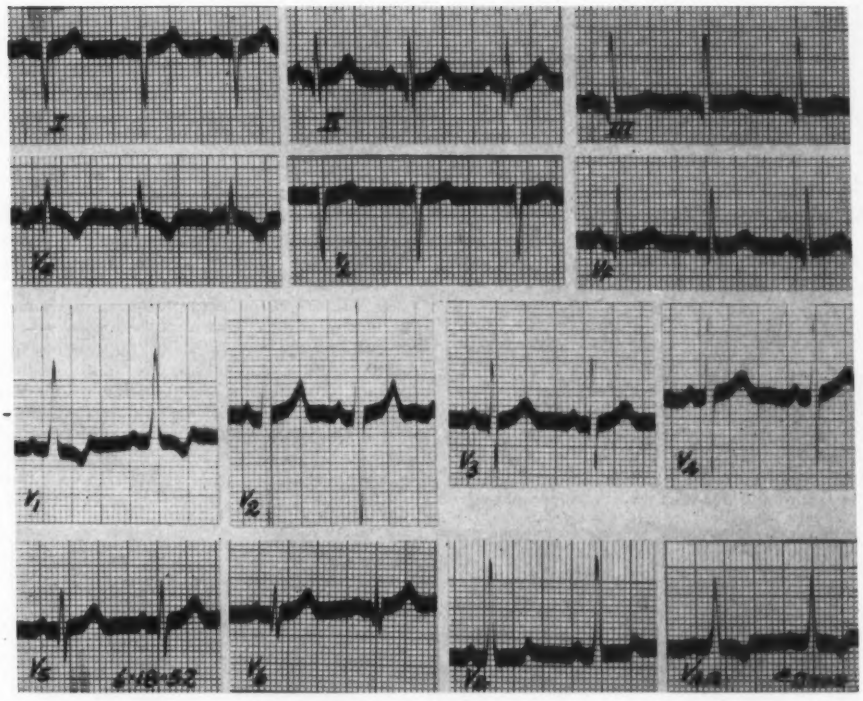

FIG. 5. Case 3. Woman, aged 24 years, with tetralogy of Fallot. Electrocardiograms recorded during the fourth month of a first pregnancy show classic changes of pronounced right ventricular hypertrophy.

of pregnancy after the Blalock procedure have been recorded by Russell and his associates[18] and by Olim and Turner.[16] The following case illustrates a successful pregnancy in this disorder without any such operative procedure.

CASE 3.—M. P., aged 24 years, was first seen June 18, 1952, during the fourth month of her first pregnancy. Cyanotic heart disease had been recognized since infancy. Although unable to participate in sports of any kind, the patient had been able to attend grammar school and to carry out light household duties thereafter. The clinical features were characteristic of tetralogy of Fallot and electrocardiograms suggested pronounced right ventricular hypertrophy (fig. 5 and 6).

The entire pregnancy was uneventful. Cyanosis of moderate degree was present throughout, but it did not increase in degree. The vital capacity never changed appreciably from the 2,600 cc. recorded on the initial examination. A normal infant was delivered vaginally at full term with no complications.

Some concern was felt about the infant in this case, since Olim and Turner[16] reported anencephaly in 2 fetuses of a mother with tetralogy of Fallot only a short time after my patient was seen initially. In this report attention was called to the possibility that the low oxygen tension in the mother's blood may affect the cephalic phase of development adversely, drawing a parallel with the experimental production of anomalies by maternal insult in the form of anoxic anoxia. Fortunately, my patient was delivered of a normal infant. In a series of more than 10 patients with congenital heart disease, followed through pregnancy in

FIG. 6. Case 3. Roentgenographic study of the heart shows cardiac configuration of surprisingly normal contour even though the patient was distinctly cyanotic and had electrocardiographic evidence of right ventricular hypertrophy. A normal fetus was born at full term after uncomplicated pregnancy.

the past six years, only a single abnormal fetus was born of an elderly primipara with a patent ductus arteriosus but no evidence of cyanosis.

A new problem in this field is the management of patients with mitral stenosis who have successful valvulotomy and become pregnant thereafter. Massey[13] indicates that at least a score of such patients have come to his attention. He also mentions a successful commissurotomy performed by Rumel during pregnancy. This procedure also has been accomplished by Baker, Brock, and Campbell[1] and by Logan and Turner.[12] Such a case is described briefly through the courtesy of Dr. Donald M. Willson.

CASE 4.—A. P., aged 32 years at the time of delivery, had had rheumatic fever at 17 years of age. The mitral stenosis which resulted limited her activities severely. Congestive heart failure and auricular fibrillation developed in 1950. In November, 1950, the patient had an embolus to the right popliteal artery (fig. 7). A mitral commissurotomy performed by Dr. Charles Bailey in January, 1951, was followed by a striking improvement in function so that the patient undertook all normal activities. The auricular fibrillation present prior to operation persisted unchanged. The patient became pregnant one year after the operation and delivered a normal infant after an entirely uneventful pregnancy.

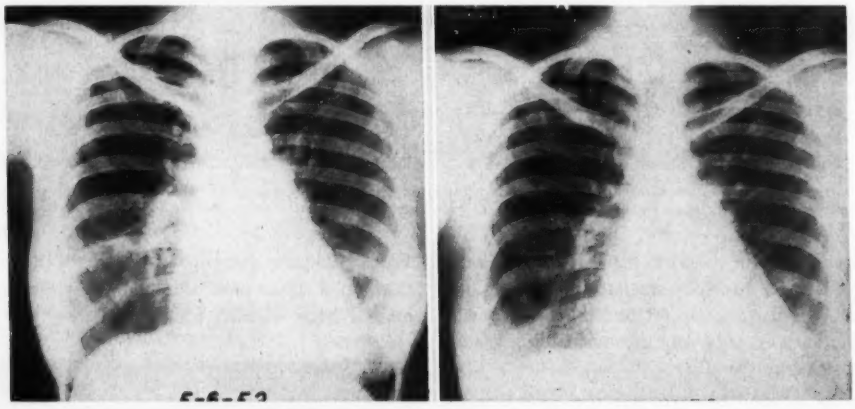

FIG. 7. Case 4. Woman, aged 32 years, with mitral stenosis and successful mitral commissurotomy performed in January, 1951. *Left.* Roentgenographic study of the heart during pregnancy, which began slightly more than one year after commissurotomy. *Right.* Roentgenographic study of the heart at full term shows mitral configuration and apparent slight increase in the size of the heart as compared with the earlier roentgenogram. A normal fetus was born at full term after an uncomplicated pregnancy.

More and more of these patients will present themselves in the future. The criteria for the risk of pregnancy must be altered in these women because the structural valvular disorder has been altered. Evaluation must be based upon the postoperative functional capacity. It is probably wise that such patients wait at least one year after operation for full benefit to develop and to permit a proper evaluation by the physician before pregnancy is undertaken.

In 1951, McRoberts[14] reported several cases in which the dorsal recumbent position during pregnancy produced discomfort and circulatory collapse. The following case seems related to this situation and is reported as an illustration of the type of problem which often causes the obstetrician some concern.

CASE 5.—E. D., aged 22 years, was seen first March 9, 1949, during the eighth month of her first pregnancy. One month earlier when placed in the dorsal recumbent position in the office of her obstetrician she had become faint and dyspneic, and tachycardia had appeared. The symptoms cleared promptly when she sat up.

The sensations recurred one month later under the same circumstances. On three separate occasions during March, 1949, the heart rate was observed to increase from levels of 75 to 88 per minute on first assuming the dorsal recumbent position to 152 to 164 per minute within three to seven minutes (fig. 8). The rate returned abruptly to normal levels when the patient turned to the left lateral position. However, at the time of labor the phenomenon was less pronounced and delivery was carried out in the usual way.

The patient is now in the course of a second pregnancy. During the eighth month of this pregnancy, the heart rate rose from 84 to 114 per minute while she lay flat on the examining table.

The explanation for such cases is obscure. McRoberts has suggested that obstruction of venous return from the lower extremities results from pressure by the pregnant uterus with the patient in the supine position. The resultant fall in right auricular pressure then results in a decreased cardiac output. If this hypothesis is true, it should be possible to reproduce the symptoms by application of venous tourniquets to the thighs even with the patient on her side. I have not had the opportunity to make this observation.

MANAGEMENT OF LABOR

The actual management of labor in patients with heart disease need not be dealt with in detail except

to point out a few important principles. With the exception of coarctation of the aorta, cesarean section should be performed in patients with heart disease solely for obstetric indications. The choice of the anesthetist is more important than the choice of anesthesia, although the agent should be one which permits a fairly high concentration of oxygen and which is not prone to excite cardiac arrhythmias. I have a personal preference for ether anesthesia in these patients.

The second stage of labor should be shortened judiciously. Some years ago Barker[2] suggested the application of venous tourniquets to all four extremities after delivery of the placenta. I have adopted this practice with the thought that contraction of the uterus forces blood into the general circulation abrupt-

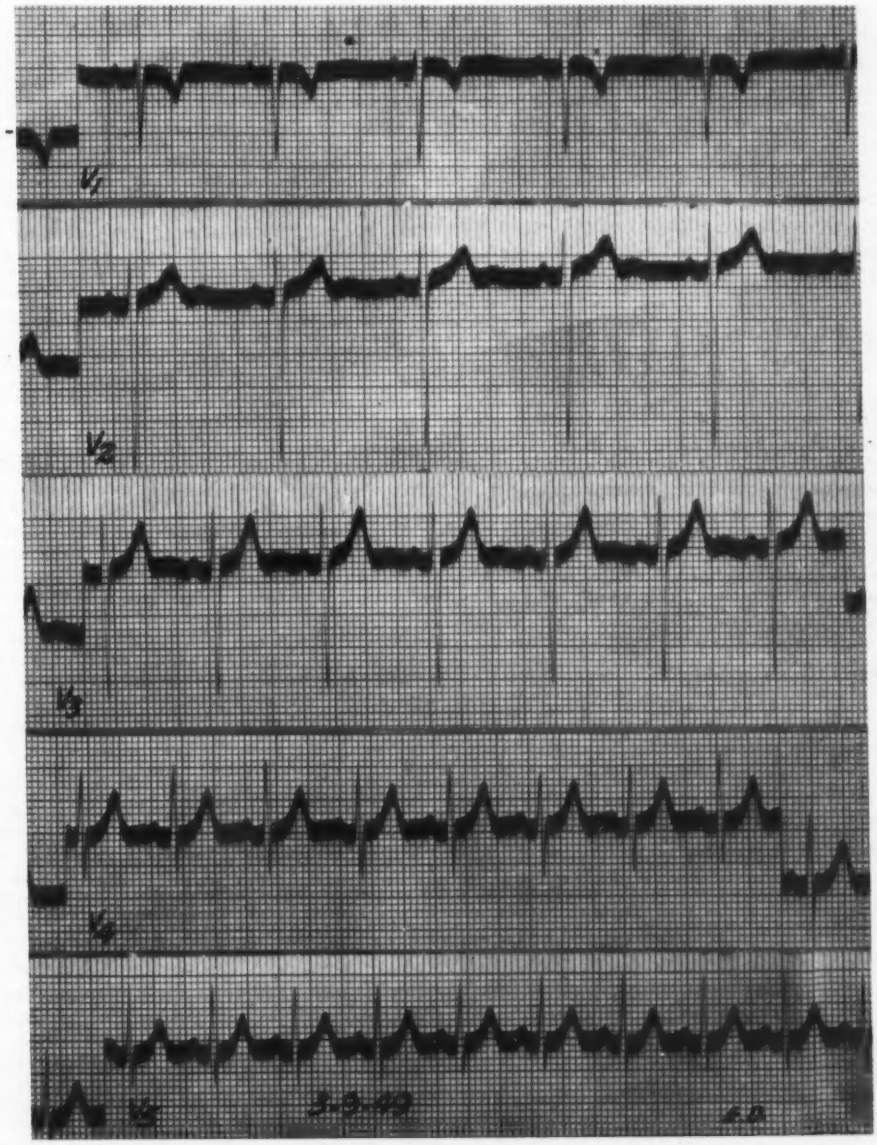

FIG. 8. Case 5. Woman, aged 22 years. Electrocardiographic observations were made during the eighth month of a first pregnancy. Marked sinus tachycardia was observed repeatedly when the patient assumed the dorsal recumbent position. Records shown were taken in rapid succession with the patient lying on her back. Lead V_1 shows a ventricular rate of 66 per minute. Subsequent records show progressive increase in the heart rate to 116 per minute in lead V_5. At times the rate rose to 164 when the patient assumed this position.

ly, and by exclusion of a large volume of blood in the extremities for one or two hours patients may be carried through a potentially dangerous, immediate postpartum period.

Prophylaxis with antibiotic agents against the possibility of subacute bacterial endocarditis has been my practice, and in my modest series of patients this complication has not occurred. Penicillin usually is given for five to seven days after delivery.

Most observers are agreed that early ambulation has no place in the treatment of cardiac patients.[8, 13] It must be remembered that there is a rebound in the load of pregnancy following delivery (fig. 1), and the postpartum period is also a hazard to these patients.

Fetal mortality in mothers with heart disease tends to be high. Although only slightly higher than in normal mothers in uncomplicated pregnancies, the rate may be as high as 30 per cent when congestive failure occurs.

CONTRAINDICATIONS TO PREGNANCY

The contraindications to pregnancy are those features which aggravate the risk of pregnancy far beyond the average. They include a past or present history of congestive failure, acute rheumatic fever, failure appearing in the first trimester, and most instances of auricular fibrillation. Economic, religious, and social factors must always be taken into account. If a pregnancy is terminated, this should be accomplished before the fifth month because a hysterotomy after that date may prove more hazardous and complicated than delivery at term.

SUMMARY

The current understanding of the physiologic load of pregnancy has made more intelligent the physician's approach to the cardiac patient who becomes pregnant. New advances in the treatment of heart disease have brought new problems in this special field because patients who could not previously withstand the load of pregnancy are now being made able to do so. The close cooperation of the obstetrician and the internist or cardiologist has become even more important in the proper management of these cases.

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ABSTRACT OF DISCUSSION

DR. E. A. CHANDLER, Houston: Dr. Rosenbaum has outlined the latest methods of management of heart disease in pregnancy, and it behooves all of us to use his teachings.

More than fifteen years ago, when I first began seriously to study heart disease in pregnancy, less was known than today and there was little agreement as to the proper management of these cases. Now there is a better understanding and knowledge of heart disease, and more definite agreement on conduct of these patients through the various stages of pregnancy and delivery. Treatment of rheumatic heart disease has improved, more knowledge has been gained about the physiologic changes during pregnancy, and much valuable cardiovascular surgery has been developed. All of these factors have been of great benefit.

Most of the mortality of heart disease in pregnancy has been caused by lack of cooperation (patients' fault), and lack of ability to evaluate and predict the outcome of each case and lack of proper attention to patients during labor and the immediate postpartum period (doctors' fault). In order to improve mortality rates physicians must continue to educate the public and themselves and to practice eternal vigilance in the conduct of pregnancy and labor.

ESSAY CONTEST ON CHEST DISEASE

The American College of Chest Physicians will present three awards for the best original contribution, prepared by any student in an accredited medical school, on any phase of the diagnosis and treatment of chest disease. Each winner will be given a certificate of merit, and the first place winner will also receive a cash award of $250 at the twentieth annual meeting of the college in San Francisco June 17-20.

Deadline for the manuscript to be submitted to the executive director of the college, 112 East Chestnut Street, Chicago 11, is March 15.

TOBACCO ADDICTION
A Suggestion as to Its Remedy

CHARLES T. BROWN, Lt. Col., MC, USA, Fort Sam Houston, Texas

MARK TWAIN once remarked: "It is easy to give up tobacco; I have quit a thousand times." The humor in his statement cannot be denied; however, it seriously reflects the experience of the older smoker who has spent a good part of a lifetime in the consumption of more than one-half million cigarettes. Such a person is in a good position to elaborate on literally hundreds of attempts on his part to abandon the poison threatening his very existence. Similar unsuccessful attempts by millions of other tobacco users since the time of Raleigh would indicate once more that the substance under discussion is not to be dealt with lightly. The user of tobacco who wishes to refute the addicting qualities of this drug has only honestly to examine his own experience.

TOBACCO: A NARCOTIC

Tobacco is a narcotic capable of producing tolerance, dependency, and withdrawal phenomena in those persons accustomed to its use. Thus, it fulfills the requirements for the definition of an addicting substance. The skeptic who is unwilling to accept this fact will find solace in the words of Sollmann,[2] who writes of the toxic effects of the active principle of tobacco, the alkaloid nicotine:

> Nicotine is one of the most fatal and rapid of poisons; the vapors arising from a glass rod moistened with it and brought near the beak of a small bird causes it to drop dead at once, and 2 drops placed on the gums of a dog may give a similar result. . . . It acts with a swiftness equalled only by hydrocyanic acid.

Aside from its addicting properties, there appears to be ample evidence that there is a relationship between tobacco and physical disease. Carcinoma of the lung, coronary and peripheral vascular disease, nutritional and nervous disorders, peptic ulcer—all are the vade mecum of nicotine, and their unnatural alliance cannot be denied or ignored.

It is a strange commentary on the public at large, and especially on the medical profession as a class, that despite repeated and pointed warnings in both lay and professional literature of the deleterious and even fatal implications of tobacco addiction, use of the drug continues in rising proportion. This has been the history of other narcotics. The extensive use of opium and its derivatives began in this country just prior to the Civil War and continued to spread unabated until its curtailment by the enactment of the Harrison Narcotic Act in 1915. Another narcotic, barbituric acid, first synthesized by Fischer in 1903, after half a century became known as a dangerous substance with addicting properties and other lethal potentialities. Cannabis, popularly known as marihuana, was not classed as a narcotic until the passage of the Marihuana Tax Act in 1938. Similar legislation and restrictions finally were applied to meperidine and other synthetics and later to mescaline and peyote. Another narcotic, alcohol, needs no comment here.

Certainly, tobacco and heroin should not be placed in the same company; however, under certain conditions a .22 rifle has been known to possess all the lethal potentialities of a .50 caliber machine gun. The man dead of carcinoma of the lung after years of heavy cigarette smoking and his brother whose death is the result of heroin deprival may well be from a standpoint of toxicology in *status quo*. The essential differences in the causative agents of their demise can be argued and delineated from a standpoint of pharmacology by professors in the classroom; nevertheless, the fact remains that the agents of their deaths could well be the addicting drugs of their respective choice. And as Metchnikoff said: "Man does not die; he poisons himself."

The question of motivation is paramount in the determination of any form of behavior. The person who desires to achieve worth-while goals must be prepared to make certain sacrifices ranging from minimal to heroic, depending upon the nature and magnitude of his undertaking. The renunciation of tobacco for some is a simple task; for others the task is of Herculean proportion. Unfortunately, the work to be done often is rendered no less easy by news of a heart disease or a suspicious lung lesion that portends catastrophe. The scheme that follows is not for the strong who wear their chains lightly; rather, it is for the miserable slave whose shackles bruise and grow heavier with the passing of the years.

SUGGESTED REMEDY

Some 200 years ago, a venerable apothecary of London whose name has been lost in obscurity, becoming alarmed over the increasing prevalence of "snuff taking" and its deleterious consequences, offered a remedy for those afflicted with what he considered to be an unwholesome pastime. It is the purpose of this

From the Department of Neuropsychiatry, Medical Field Service School, Brooke Army Medical Center.

paper to present the proposed remedy as a simple and effective approach to the abandonment of tobacco. The plan is unique in that it paradoxically allows the tobacco addict to continue the use of the drug while in the process of its complete renunciation. The plan of treatment is as follows:

The patient proceeds upon a planned schedule with the determination to adhere faithfully to its simple provisions. The principle involved is the progressive reduction of the tobacco intake by one hour each day. The subject, for example, arises at 6 a. m. and will thus embark upon his reduction at that hour for the first day. He starts smoking at 6 a. m. and may smoke all that he chooses until midnight, or thereabouts, when he normally retires for the night. The next morning, or the second day of the treatment plan, he moves his smoking time up one hour to 7 a. m. Again, he may smoke all that he desires until midnight. On the third day he moves his smoking activities up until 8 a. m., and so on successively to the end that smoking is delayed by one hour each day until the subject reaches midnight of the final day. He is now aware of the fact that during the eighteen day period he has progressively diminished his smoking time by one hour each day and finds that on the last day he has only one hour, from 11 p. m. until midnight, to indulge in smoking. Again, he may smoke all he wishes during this final hour. At its conclusion, he retires to bed having completed the reduction treatment.

COMMENT

Obviously, the treatment as outlined in this presentation is no more than a gradual reduction procedure with the goal of detoxification common to all drug withdrawal plans. However, this plan is more than that and upon this hinges the secret of its success. In the case of tobacco, as with other narcotics, there are both psychologic and physiologic factors in addiction.

In consideration of one of the more important psychologic components of the problem, it is a well known fact that a major obstacle in the withdrawal of any narcotic is the patient's frightening prospect of an existence without support of the drug. In other conventional forms of narcotic drug withdrawals such a prospect exists throughout the treatment. However, in the plan proposed the patient is assured positively from the first day that he not only will be allowed to smoke, but will further be permitted to smoke as much as he chooses. He is required only to delay for a relatively short, though increasing, time each day his initial indulgence in tobacco. The positive assurance of unlimited smoking privileges is sufficient to sustain him through the progressively increasing period of daily abstinence.

As to the physiologic component of tobacco withdrawal, standard references on pharmacology give a detailed account of the altered physiology of each organic system incident to the ingestion of tobacco. Obviously, as the subject limits and decreases his daily intake of the narcotic and his exposure to the drug is lessened progressively each day, there will be a gradual restoration of normal bodily functions. Along with this gradual restoration of bodily health there is uniformly an abatement of the abstinence symptoms, both psychologic and physiologic, to the end that the patient has achieved a psychic and somatic tranquillity heretofore unknown in a tobacco laden atmosphere.

The question now arises as to the possibility of relapse. Experience shows the possibility of such an eventuality, for relapse in drug addictions is the rule rather than the exception. According to Otto Fenichel,[1] addicts are persons who have a disposition to react to the effects of drugs in a specific way, namely, in such a way that they try to use these effects to satisfy the archaic oral longing which is sexual longing, a need for security, and a need for the maintenance of self-esteem simultaneously. Thus, the origin and the nature of the addiction are not determined by the chemical effect of the drug, but by the psychologic structure of the patient. So the premorbid personality of the subject is the decisive factor, both as to the initial exposure to tobacco and as to the continuing use of the drug. It may be postulated that so long as tobacco fulfills some inner need or gratification to the subject or satisfies again the archaic oral longing, he will be perpetually in danger of relapse. However, one or more relapses should not be so discouraging to the patient as to persuade him that his situation is hopeless. One, or even a succession of relapses, does not necessarily mitigate against ultimate and complete success with its reward of permanent and irrevocable renunciation.

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SURGEONS TO HOLD SECTIONAL MEETINGS

The medical profession at large is invited to attend any of six sectional meetings to be held by the American College of Surgeons during the first six months of 1954. Panels, symposiums, papers, and motion pictures will be presented at each session, which will concentrate on problems encountered in day-to-day practice. The meetings are scheduled as follows: February 1-3, Charlotte, N. C.; February 25-26, Reno, Nev.; March 1-4, Omaha, Neb.; March 15-17, French Lick Springs, Ind.; March 31-April 2, Montreal, Quebec, Canada; May 17-19, London, England.

Details may be obtained from Dr. H. Prather Saunders, associate director of the American College of Surgeons, 40 East Erie Street, Chicago 11.

CASE REPORTS

ENDOCARDIAL SCLEROSIS

WILLIAM L. HOWELL, M.D., Washington, D.C.

ENDOCARDIAL sclerosis, by definition, is a fibrous thickening of the endocardium. Sclerosis of the valvular endocardium is viewed with suspicion, and when discovered, it is routinely studied microscopically by pathologists. Sclerosis of the mural endocardium, however, has received scant attention, except as a striking feature of "fibroelastosis," a uniformly fatal entity occurring in infants. This is understandable as endocardial sclerosis is often an incidental finding in diseased hearts. White plaques of endocardial thickening are frequently seen overlying healed myocardial infarcts. In coronary sclerosis without infarction, arterial hypertension, valvular heart disease, congenital anomalies of the heart and coronary circulation, chronic lung disease producing pulmonary hypertension, and isolated myocarditis, more diffuse thickening is sometimes seen. Sclerosis of the mural endocardium, however, may occur in the absence of other demonstrable heart disease and be the only abnormal anatomic finding other than cardiac hypertrophy in certain patients dying of congestive heart failure.

These patients are usually young adults who develop advanced congestive heart failure within a few weeks; their previous history is asymptomatic. Clinically, these patients exhibit cardiomegaly and gallop rhythm, but show none of the stigmas usually associated with common forms of heart disease. The blood pressure is often low; retinal vessels are normal. No significant murmurs are found. The temperature, white blood cell counts, and sedimentation rates are normal. Tremors, eye signs, thyroid enlargement, and quadriceps weakness, which might indicate hyperthyroidism, are not present. There are no signs of myxedema. Roentgenograms show cardiac contours of generalized dilatation without suggestion of hypertensive, valvular, or congenital heart disease. They resemble hearts made abnormal by beriberi, but there is no response to therapy with thiamine chloride. Sooner or later embolic phenomena occur in most cases, leading to a suspicion of subacute bacterial endocarditis. Incident to infarction, fever, leukocytosis, and an elevation of the sedimentation rate strengthen this suspicion. But repeated blood cultures are negative. A poor response to digitalis is mentioned in almost all cases reported. Mercurial diuretics, cation-exchange resins, and low salt diets may help temporarily, but the course of the disease is progressively downhill. Death usually occurs within a few months to a year.

Two cases of endocardial sclerosis are presented in this report. The clinical and pathologic features of the condition are discussed.

Read before the Section on Internal Medicine, Texas Medical Association, Annual Session, Houston, April 28, 1953.

CASE REPORTS

CASE 1.—A 41 year old meat department supervisor was admitted to the hospital September 16, 1952. He complained of shortness of breath of three weeks' duration.

In 1945, early in the course of surgery for an inguinal hernia, the patient's heart ceased to beat for a short time and the incision was closed. The operation was completed successfully ten days later. His past history was otherwise negative.

The patient first noted dyspnea when he lifted a meat basket that weighed approximately 25 pounds. The following day, walking a distance of only 50 feet made him breathless. One week prior to hospitalization he had been confined to bed; on several occasions he had been forced to sit upright to breathe comfortably. On the day of admission he coughed up bright red blood.

Physical examination revealed a dyspneic, well nourished white male about 40 years of age, in bed with the head elevated approximately 45 degrees. The patient's temperature was normal, the pulse rate was 140 per minute, and respirations 30 per minute; blood pressure was 106/70. Rales were present at both lung bases. A diastolic gallop was present at the apex, but there were no murmurs. The liver edge was palpable at the umbilicus, and there was extreme tenderness in the right upper quadrant of the abdomen. There was no peripheral edema.

An electrocardiogram revealed sinus tachycardia and a left ventricular strain pattern.

A roentgenogram of the chest showed an enlarged heart and evidence of pulmonary congestion.

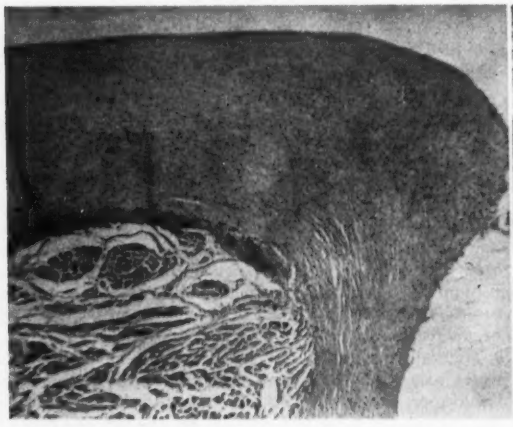
The patient was given digitalis, oxygen, a mercurial diuretic, and morphine sulfate, and was moderately improved within twelve hours. The pulse rate had slowed to 90, but in another twenty-four hours it had become rapid again; it was rarely below 120 per minute the remaining three months of his life despite full digitalization. Follow-up roentgenograms revealed a large pulmonary infarct to be the source of his hemoptysis. Anticoagulant therapy was instituted.

The course remained largely afebrile, and white blood cell counts and sedimentation rates were normal. Slight elevations of temperature followed embolic episodes; there were many of these. An attack of flank pain associated with microscopic hematuria, after anticoagulants had been discontinued, was interpreted as being due to renal infarction. It was thought a short period of aphasia was the result of cerebral em-

bolization. On at least four occasions it was possible to make a positive diagnosis of recent pulmonary infarction.

Repeated blood cultures were negative. As one of the several studies of peripheral blood was thought to be positive for "L. E. cells," cortisone was used in daily doses of 200 to 400 mg. for eight days without benefit. During the last month of the patient's illness, changing systolic murmurs appeared and splinter hemorrhages were noted beneath the nails. These findings suggested subacute bacterial endocarditis. Thus, penicillin, and later Terramycin were used, without benefit. Death occurred suddenly three and one-half months from the onset of his illness.

At autopsy, fluid was present in all serous cavities in moderate amounts. The lungs contained multiple infarcts which showed various degrees of organization. Antemortem clots of various sizes were present in many branches of the pulmonary arteries. The heart was greatly dilated and weighed 500 Gm. White plaques of endocardial thickening were present in the apices of both ventricles. Within the cavity of the left ventricle an ovoid-shaped organized thrombus, measuring 8 by 4 by 3 cm. was firmly attached near the apex.



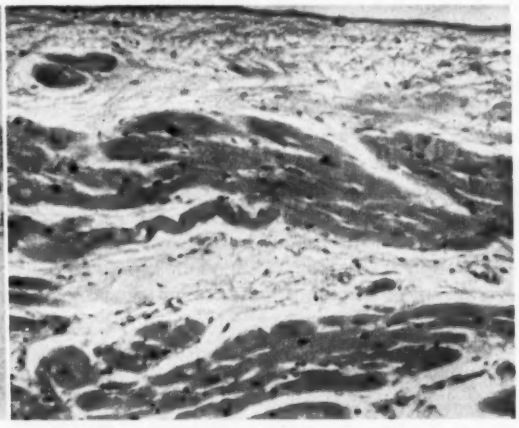


FIG. 1. Left. Photomicrograph showing thickened endocardium overlying the papillary muscle.

Right. Photomicrograph showing the extension of fibrosis into the subendocardial myocardium.

Smaller organized thrombi were attached at the apex of the right ventricle and in the right auricular appendage. Grossly, the valves and coronary vessels appeared normal. Both kidneys contained recent and healed infarcts.

Microscopically, the thickened endocardium and focal areas of fibrosis in the subjacent myocardium were striking. Mild scattered round cell infiltration was present. The coronary vessels showed minimum eccentric intimal thickening.

CASE 2 (Courtesy of Col. T. S. Mattingly, U.S.A.).—A 38 year old white man was admitted to the hospital June 14, 1949, with a chief complaint of shortness of breath and swelling of the ankles of three weeks' duration.

In 1945, because of pain in the region of the umbilicus, an appendectomy was attempted, but when the abdomen was opened, heart and respirations ceased. The patient was revived and the abdomen closed; the appendix was not removed at this time. In 1948, he was admitted to the hospital twice because of abdominal pain. In one instance a perforated duodenal ulcer was suspected, but a cardiologist was called when a fast heart rate was noted. Supraventricular tachycardia was confirmed by electrocardiogram, and an acutely distended liver subsided after treatment.

Following the last-mentioned episode, which occurred in November, 1948, the patient remained asymptomatic until three weeks before his admission in June, 1949. For one week he had suffered attacks of nocturnal dyspnea.

Physical examination revealed a well developed white man with dusky cyanosis of lips and extremities. The temperature was normal; the pulse rate was 116 per minute and the rhythm was regular. The point of maximal impulse was in the anterior axillary line in the sixth interspace. There was 3 plus edema of the lower legs.

A complete blood count and sedimentation rate were normal.

Roentgenograms of the chest revealed an enlarged heart without characteristic contour.

An electrocardiogram showed sinus tachycardia, left axis deviation, and abnormal P waves.

The patient was placed in an oxygen tent as he was cyanotic, and was rapidly digitalized. On the morning of the third day the pulse rate was recorded by the nurse as 82 per minute. Shortly thereafter he suddenly became cyanotic and expired.

Autopsy revealed 1,500 cc. of clear fluid in the right pleural cavity and 500 cc. in the left pleural cavity. The heart weighed 675 Gm. Both ventricles were dilated. The endocardium was moderately to markedly thickened, and organized thrombi were firmly attached to the apex of the left ventricle. The valves were normal. The coronary arteries were not unusual. Other findings included passive congestion of liver and lungs, and a recent infarct of the spleen.

Microscopic examination revealed endocardial sclerosis, focal fibrosis of subjacent myocardium, and scattered round cell infiltration.

DISCUSSION

Thickening of the endocardium received scant attention until the report of Bedford and Konstam[1] in 1946. They described cardiac dilatation, fibrous thickening of the endocardium, and focal fibrosis in the subjacent myocardium as the only significant finding in 40 young West African soldiers dying of congestive heart failure. Two years later, Davies[3] reported similar findings in East African natives and called the condition endocardial fibrosis. Ian Gray[4] in London in

1951, reporting the condition in Europeans, pointed out that whereas cases in Africa might be due to some obscure infestation or infection, the same gross pathologic picture had been described in the medical literature under a variety of titles. These included "cardiac hypertrophy of unknown etiology," "chronic fibroplastic myocarditis," "thromboendocarditis," and others. He referred specifically to cases reported by Levy and Rousselot,[6] Reisinger and Blumenthal,[10] Löffler,[7] and Ware and Chapman.[11] Recently similar cases have been reported by MacMahan, McNicol, Benenson, and Winship,[9] and one from the records of Massachusetts General Hospital.[8]

In cases of endocardial sclerosis, gross specimens show hearts that are noticeably dilated. Actual hypertrophy not so apparent upon inspection is evidenced by weights of 500 and 600 Gm. Sclerosis of the endocardium may occur in white plaques of irregular shape, or it may be diffuse. It is usually most evident near the apices of the ventricles. The entire parietal endocardium may be involved. Organized mural thrombi occur most frequently near the apices of the ventricles, where they extend into the bays between papillary muscles to attach themselves firmly; they may become so large they diminish ventricular filling. Other thrombi may be found in the auricles or their appendages. Cut surfaces of ventricular walls may or may not show subendocardial areas of focal fibrosis. The heart valves, coronary vessels, and pericardium appear normal.

Microscopically, the endocardium is many times its usual one-cell thickness. The subjacent myocardium may show focal areas of fibrosis, or these areas may be elsewhere in the myocardium. They do not have a significant relationship to blood vessels, and they do not occur in all cases. There may be some increase in interstitial stroma. In some areas individual myocardial fibers may be separated, some may be hypertrophied, and some seemingly have undergone pressure atrophy. This pathologic picture has been given the name "serous myocarditis." Small vessels in the subendocardium may contain thrombi. A scattering of round cells may be observed, but careful search fails to reveal focal cellular infiltration as seen in isolated myocarditis.

There are various theories concerning the etiology of endocardial sclerosis: (1) that it results from a dietary deficiency, (2) that it is residual of isolated myocarditis, or (3) that it is a primary chronic parietal endocarditis. Bedford and Konstam, in their original report, suspected a dietary deficiency but found that their patients did not respond to large doses of thiamine chloride. Ian Gray thought that this report excluded a vitamin deficiency as an etiologic factor since heart failure due to thiamine deficiency usually responds promptly to treatment; furthermore, it is the only vitamin deficiency known to produce heart failure. He pointed out that Chagas' disease, or South American trypanosomiasis, produces sclerosis of the endocardium, which supports the opinion that other infections or infestations might likewise produce it. Dammin[2] has reported fibrous thickening of the endocardium, focal fibrosis of myocardium, and active focal myocarditis in the same specimen. One explanation for the cause of endocardial thickening in association with focal myocarditis is that focal myocarditis interferes with blood supply. Finally, the hypothesis that endocardial sclerosis is a primary condition affecting the endocardium does not explain focal fibrosis and other changes in the myocardium. Although Gross[5] explained focal fibrosis as being caused by occlusion of thebesian, arterioluminal, and arteriosinusoidal channels, Wiggers has not found these to be functional in the presence of normal hemodynamics.

SUMMARY

Sclerosis of the mural endocardium may be the only abnormal anatomic finding, other than cardiac hypertrophy, in certain young adults dying of congestive heart failure.

Two such cases are presented.

Clinical features, pathology, and theories as to the etiology of the condition are discussed.

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ABSTRACT OF DISCUSSION

DR. GEORGE R. HERRMANN, Galveston: I have seen the hearts of 3 infants with endocardial fibroelastosis, 2 of which were reported by Dr. C. Ralph Letteer of San Antonio in the March, 1953, *Annals of Internal Medicine*. These cases

were recognized from the clinical data by Dr. Louis Manhoff. The other juvenile was not recognized in Galveston but went to postmortem with the diagnosis of congestive heart failure and cardiac enlargement of unknown origin.

I saw 1 adult case in an officer at Brooke Army Medical Center in San Antonio. The officer had returned from Korea and was doing special work at Fort Leavenworth when symptoms of heart failure suddenly began and progressed rapidly. He was transferred to Fort Sam Houston for care, diagnosis, and treatment. He had many of the symptoms which Dr. Howell has outlined, and most of the suggested erroneous diagnoses were made. I thought that he had a hypoplastic aorta, but particularly a hypoplastic coronary artery system. Col. R. B. Johnson agreed, but Col. Carl J. Lind, the pathologist, returned the diagnosis of endocardial fibroelastosis; however, he has not yet made his final report.

Dr. Howell has covered adequately this unusual and probably frequently overlooked condition; I would only add coronary hypoplasia among the theories as to the etiology of endocardial fibroelastosis. We probably make the common error of misdiagnosing some of these cases in which no cause has been apparent as chronic and diffuse coronary artery heart disease, which seems to explain the picture. On several occasions in clinical pictures of this type I have found evidence of a narrow great arch and made the diagnosis of hypoplastic aorta and coronary artery system. I was made aware of this syndrome of hypoplasia of the aorta by one of my oldest students, G. Werley, who died in El Paso recently. He sent me a patient with the diagnosis of hypoplastic aorta twenty years ago. The diagnosis was confirmed by Mavis Kelsey, then a member of the Department of Pathology at the University of Texas Medical Branch, who made a careful study of the case. He also studied the caliber of the aortas and recorded that the hypoplastic aorta and hypoplastic coronary arteries may have given off at right angles extremely hypoplastic muscular branches. The gradient of blood pressure drops off sharply in such arterial systems. This results in decreased blood flow in the transmural branches that run off at right angles from the epicardium almost to the endocardium. This could easily account for the endocardial ischemia and injury and later fibrosis.

The electrocardiograms are suggestively characteristic, I believe, in most of the cases (as I pointed out to Dr. Letteer in his first case and noted in Dr. Howell's first case before digitalization), a depression of the ST segments and negative T waves in leads I, II, III, V_4, V_5, and V_6 indicating an injury and ischemia in the subendocardial tissue. I have always insisted upon a description of the RST changes rather than using the term ventricular strain, which term should be abandoned. Certainly in these instances the blood pressure is low and one cannot visualize physical strain. It is much more logical to visualize a subendocardial ischemia and injury from coronary insufficiency or even digitalization.

Subendocardial fibroelastosis may not be far removed from sclerodermatous heart disease. Dr. Howell has dealt with the collagen disease etiology as a possibility. I have seen the hearts of patients with Chagas' disease at the Oswaldo Cruz Institute in Rio de Janiero and noted that the myocardial process is much more diffuse. Triatoms infected with Trypanosoma cruzi are found in Texas; we should have Chagas' disease myocarditis in Texas, but we have not recognized it.

ACUTE HEMOLYTIC ANEMIA
Report of 3 Cases in One Family

J. LEIGHTON GREEN, M.D., El Paso, Texas

FAMILIAL hemolytic anemia, also known as spherocytic anemia, chronic hemolytic jaundice, or acholuric jaundice, is a disease characterized by chronic or recurring anemia associated with hemolytic crises, increase in fragility of the erythrocytes, a tendency for the red cells to be spherical and microcytic, high reticulocyte count, increased excretion of urobilin in the urine, splenomegaly, and usually icterus. Doan said that "true hemolytic icterus is always the manifestation of an inherited constitutional defect." However, there is also a so-called acquired type, which most often manifests itself later in life than the congenital form. The acquired form is usually the more severe.

Hemolytic anemia seldom becomes apparent in infancy, but is usually detected before adolescence. Pallor and slight jaundice may be the first signs, together with a mild anemia. Usually the spleen is enlarged. First indication of the disease may be a crisis.

Hemolytic crises may be mild or they may be fatal. A crisis may be precipitated by trivial illness, emotional stress, or fatigue. Crises occur at varying intervals, with fever, abdominal pain, pallor, jaundice, and weakness. During a crisis there is excessive destruction of red blood cells, as shown by a lower red cell count, rising icteric index, and increased urobilin in the stools and urine. The urine is highly colored. The stools appear normal in color and consistency, but contain much urobilin. There is always a high reticulocyte count, perhaps to 95 per cent. Fragility of red blood cells to hypotonic sodium chloride solution is noticeably increased, as a rule. The white cell count is not characteristic but most frequently shows a leukocytosis, with increase of polymorphonuclear leukocytes. It has been shown that hepatic function is diminished during a crisis, a form of hepatitis being present. Cholecystitis is commonly found associated with the anemia, and cholelithiasis is frequent, even in the very young.

Splenectomy is curative. It usually relieves all symptoms; however, the increased fragility of red blood cells may persist long after the operation. It is best

not to perform the operation during an acute crisis, but when the crisis is unduly prolonged, surgery may be indicated. Transfusions may be harmful during a crisis but are often necessary as a preoperative measure when the blood count is extremely low.

This report concerns a family in which hemolytic anemia has appeared in three successive generations. I have performed splenectomy on three members of the family. One grandfather is said to have died of anemia, without benefit of surgery. His daughter (case 2) was relieved by splenectomy. Two of her sisters have had splenectomy, and 2 of her 3 daughters also have undergone splenectomy (cases 1 and 3). The grandfather had 3 sons, brothers of the patient in case 2. The second of these brothers has a son, aged 6 years, who is soon to have surgery for anemia, and another son, aged 1, who has anemia.

CASE REPORTS

CASE 1.—R. M. B., aged 12 years, entered Providence Hospital on June 20, 1950. Her mother said that the child had always had trouble from "spleen disease." She had been chubby until the age of 7. For the past five years she had been sick most of the time. Attacks of abdominal pain had occurred at intervals. One doctor had given a poor prognosis because of heart disease. The patient had been under treatment in Mexico, where several blood transfusions had been given. Approximately four days before she entered the hospital, jaundice had been noticed. At about the same time, she began to complain of abdominal pain and to vomit.

Examination revealed a thin, acutely ill Mexican girl. She was deeply jaundiced and dehydrated, with temperature of 100 F., pulse of 120 per minute, and respiration of 22 per minute. All mucous membranes, sclerae, and skin showed deep jaundice. The skin was excessively dry. A loud to-and-fro murmur was heard at the apex of the heart. Blood pressure was 120/72. The spleen was markedly enlarged, extending to the level of the umbilicus, and was firm. There was generalized abdominal tenderness of moderate degree. Blood count on June 20 showed red blood cells 2,700,000 per cubic millimeter, hemoglobin 7.25 Gm. per 100 cc. (50 per cent), white blood cells 7,700 with polymorphonuclear leukocytes 79 per cent, stabs 4 per cent, lymphocytes 20 per cent, and large monocytes 1 per cent. A fragility test showed hemolysis beginning at .50 per cent sodium chloride and complete at .35 (control: beginning at .40 per cent, complete at .34). The conclusion was increased fragility of erythrocytes. The hematocrit level was 26 cc. per 100 cc. of blood. The sedimentation rate was 35 mm. per hour. Kahn and Eagle tests were negative. The urine was cherry red, slightly turbid, acid, with specific gravity of 1.015, no albumin or sugar, 24 to 32 pus cells per high power field, a few squamous epithelial cells, and much bile. On June 23, coagulation time was 5 minutes, bleeding time 1.75 minutes, and icteric index 114. A cephalin flocculation test was negative.

The diagnosis was familial hemolytic jaundice, with splenomegaly. A consulting pediatrician diagnosed rheumatic mitral valve disease without decompensation. On June 21, 300 cc. of citrated blood was given as a transfusion; on June 23, another 300 cc. Following the second transfusion there was a mild reaction, with urticaria and some edema of the eyelids.

By June 24 the blood count had improved: red blood cells 3,680,000, hemoglobin 10.5 Gm. (72.4 per cent), white blood cells 5,550, polymorphonuclear leukocytes 55 per cent, stabs 2 per cent, lymphocytes 34 per cent, large monocytes 1 per cent, and eosinophils 4 per cent. Prothrombin was 100 per cent of normal.

On June 26, splenectomy was done, through a left transverse incision. The spleen weighed 610 Gm. and measured 26 cm. in the greatest diameter. There were no abnormal adhesions about the spleen. Palpation of the gallbladder and bile ducts detected no stones. During the operation, a transfusion of 500 cc. of citrated blood was given. Convalescence was uneventful. Temperature, which had receded to normal before operation, rose to 101 F. for two days postoperatively, then remained normal. Preoperatively, this patient received vitamin K with caroid and bile salts. Postoperatively, vitamin C and protein hydrolysate were administered. On June 28 another transfusion of 300 cc. of citrated blood was given. The blood count on June 30 was red blood cells 4,900,000, hemoglobin 13.7 Gm. (94.8 per cent), and white blood cells 8,150. The wound healed by first intention. Jaundice cleared up. The patient left the hospital July 4, the eighth postoperative day.

Repeated blood counts over a period of sixteen months showed the red cell count and hemoglobin within normal limits. A recent report from Mexico tells that the patient is having serious heart trouble.

CASE 2.—Mrs. J. B., aged 31, mother of the patient in case 1, was admitted to Providence Hospital January 30, 1951, for a blood transfusion. In July, 1947, I had performed a laparotomy on this woman for ruptured tubal pregnancy. Her blood count had remained low. She had been delivered of a normal child in June, 1948 (case 3). The patient gave a history of having been jaundiced at times. A cholecystogram had revealed no calculi, though the gallbladder did not fill. It was noted in the history that 2 of the patient's sisters had undergone splenectomy.

On the patient's admission to the hospital the blood count was red blood cells 3,300,000, hemoglobin 10 Gm. (62.5 per cent), white blood cells 9,500, polymorphonuclear leukocytes 67 per cent, lymphocytes 30 per cent, and large monocytes 3 per cent. Marked anisocytosis and slight poikilocytosis of red blood cells were observed. A transfusion of 500 cc. of citrated blood was given, and the patient went home the same day. Subsequent blood counts showed progressive anemia: February 6—red blood cells 3,100,000, hemoglobin 55 per cent; February 21—red blood cells 2,640,000, hemoglobin 55 per cent.

On March 6 the patient was readmitted to Providence Hospital with a diagnosis of familial anemia. She was not acutely ill, but felt tired and weak and complained of dizziness. The spleen was palpable, moderately enlarged, and tender. The blood count March 6 showed red blood cells 3,200,000, hemoglobin 10 Gm. (62.5 per cent), white blood cells 7,000, polymorphonuclear leukocytes 52 per cent, lymphocytes 37 per cent, monocytes 4 per cent, and eosinophils 7 per cent. Kahn and Eagle tests were negative. A fragility test was inconclusive. A transfusion of 500 cc. of citrated blood was given on the day of admission. Next day, March 7, splenectomy was performed through a left transverse incision. During the operation, transfusion of 1,000 cc. of blood was given. The spleen was approximately twice normal size, weighing 778 Gm. It measured 28 by 18 cm. There were some adhesions between spleen and diaphragm. A small accessory spleen approximately 5 cm. in diameter was found in the gastrosplenic mesentery. It was removed. Palpation revealed calculi in the gallbladder, which was large

and bound with adhesions. No stones could be detected in the common duct.

Convalescence was uneventful, the patient complaining only of some pain in the left anterior cervical region (from diaphragmatic irritation). Her temperature never rose above 100.8 F. She left the hospital on March 13, six days postoperatively. The wound healed by first intention.

Subsequent blood counts showed some anemia persisting: March 19—red blood cells 3,830,000, hemoglobin 65 per cent, white blood cells 11,400; September 6—red blood cells 3,430,000, hemoglobin 78 per cent, white blood cells 8,500. However, the patient gained weight and felt well. On May 3, 1952, she was delivered of an apparently normal boy. The record shows that at that time the red blood cell count was 5,200,000, with hemoglobin of 17.5 Gm. (103 per cent), and white blood cell count of 10,900. A differential count showed polymorphonuclear leukocytes 79 per cent, stabs 8 per cent, lymphocytes 16 per cent, monocytes 3 per cent, and eosinophils 2 per cent. Urinalysis was normal, and an Eagle test negative.

This patient has refused operation for gallstones, which are apparently asymptomatic.

CASE 3.—P. B., aged 4 years, is the daughter of the patient in case 2 and the sister of the patient in case 1. She was admitted to Providence Memorial Hospital March 8, 1953, acutely ill, with abdominal pain, vomiting, chills, and fever. The mother said that the illness began suddenly March 6 with vomiting. The temperature rose abruptly to 105 F. The patient complained of abdominal pain and vomited frequently. There was a slight diarrhea. The mother noticed that the urine was brick red. Only previous illness had been attacks of tonsillitis, which always responded promptly to an injection of penicillin. The mother had noticed that the child's urine was brick red on one previous occasion, at the age of 3 years.

Examination on admission revealed a very sick child, flushed, with hot, dry skin, pulse of 140, respiratory rate of 22. The temperature spiked to 105 or 106 F. daily, fell to normal or subnormal every day (as low as 95.6 F. one day). Chills were frequent. Stools were frequent, dark, and malodorous. The abdomen was slightly distended, somewhat tympanitic. It presented slight generalized tenderness, not localized. Auscultation suggested increased peristalsis. The liver extended 2 fingerbreadths below the right costal margin. The spleen could not be palpated.

Since the diagnosis was obscure, a large amount of laboratory work was done: A blood count on March 8 showed red blood cells 3,200,000, hemoglobin 9 Gm. (53.9 per cent), white blood cells 8,900, polymorphonuclear leukocytes 72 per cent, segmented cells 68 per cent, lymphocytes 27 per cent, and monocytes 1 per cent. Marked anisocytosis was found. Two normoblasts were present among 100 cells. On March 9 a fragility test showed hemolysis beginning at .48 per cent sodium chloride and complete at .34 per cent sodium chloride. A Coombs' test was negative. March 10 the blood count was red blood cells 3,400,000 (following a transfusion of 300 cc. of blood March 9), hemoglobin 11.0 Gm. (64.7 per cent), white blood cells 9,050, polymorphonuclear leukocytes 77 per cent, segmented cells 57 per cent, lymphocytes 22 per cent, and monocytes 1 per cent. Smears showed no malaria parasites.

March 11, agglutination tests were negative for typhoid, paratyphoid, Brucella abortus, Brucella melitensis, and Proteus X19. March 12 the serum albumin-globulin ratio was reported as 1.7:1.0. On the same day stool examination revealed no parasites or ova, and no blood. Later a stool culture was negative. Blood chlorides were 105.4 milliequivalents per liter with sodium chloride 154.8 milliequivalents and potassium chloride 3.8 milliequivalents. Blood culture was negative March 13 and again April 9.

The fever did not respond to antibiotics or other therapy. Supportive treatment was given, and advisability of surgery during the crisis was debated. The diagnosis of hemolytic anemia was established.

March 16 the blood count was slightly improved: red blood cells 3,500,000, hemoglobin 10 Gm. (58.8 per cent), white blood cells 6,200, polymorphonuclear leukocytes 53 per cent, segmented cells 50 per cent, lymphocytes 42 per cent, and monocytes 3 per cent.

On March 18, for no accountable reason, the temperature fell to normal, and it remained down. The patient felt well, was up and about the ward, and ate well.

On March 24, under ether anesthesia, splenectomy was done through a left transverse incision. Before the abdominal incision was made, a polyethylene tube was inserted into a vein of the right arm. This facilitated giving fluids for several days. The spleen was found to be approximately twice normal size, somewhat lobulated at its lower border. Before the splenic pedicle was clamped, Adrenalin Chloride was given hypodermically, causing contraction of the spleen. Only a few adhesions were present, about the lower pole of the spleen and about the diaphragm. As soon as the splenic pedicle had been clamped, blood transfusion was started. The pathologist reported dilated and empty sinusoids in the spleen, the picture of hemolytic anemia. The spleen weighed 79.5 Gm.

Postoperative convalescence was smooth. On the first postoperative day, temperature rose to 102.4 F. Then it quickly receded to normal, remaining normal after the third day. The patient was sent home one week after operation, on March 31. The wound had healed by this time. The patient gained weight and has remained well since. On May 28, 1953, the red blood cell count was 4,520,000, hemoglobin 15 Gm. (88.2 per cent), and white blood cell count 4,000 with polymorphonuclear leukocytes 62 per cent, stabs 2 per cent, lymphocytes 30 per cent, and eosinophils 8 per cent. The hematologist also observed occasional nucleated erythrocytes and cells showing Howell-Jolly bodies.

SUMMARY

Familial hemolytic anemia is congenital in most cases, may appear in children or adults, and has characteristic blood findings. Crises may be severe. Operation should be deferred until a remission occurs, if possible. Splenectomy cures these patients.

Three cases are presented, all in the same family, all cured by splenectomy. Family history is reviewed, with evidence of the disease in 8 members of one family.

REFERENCES

A bibliographic list may be obtained from the author.

1501 Arizona.

A legal obstacle that originally stood in the way of the civil defense program of sending help from one state to another involved professional licenses and certificates of technical skill. Many states do not recognize the rights of doctors, engineers, and similar professional people from other states to practice within their borders. Interstate compacts have now been entered into which allow all holders of "certificates of professional skill" to function in neighboring states in time of emergency.

NEWS

COMING MEETINGS AND CLINICS

Texas Medical Association, San Antonio, May 3-5, 1954. Dr. George Turner, El Paso, Pres.; Mr. N. C. Forrester, 1801 Lamar Blvd., Austin, Executive Secy.

American Medical Association, San Francisco, June 21-25, 1954. Dr. Edward J. McCormick, Toledo, Ohio, Pres.; Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secy.

NATIONAL AND REGIONAL

American Academy of Allergy, Houston, Feb. 1-3, 1954. Dr. Ben Z. Rappaport, Chicago, Pres.; Dr. Alan G. Cazort, 1425 West 7th St., Little Rock, Secy.

American Academy of Dermatology and Syphilology. Dr. Michael H. Ebert, Chicago, Pres.; Dr. John E. Rauschkolb, 25 Prospect Ave., N. W., Cleveland 15, Secy.

American Academy of General Practice, Cleveland, March 22-25, 1954. Dr. U. R. Bryner, Salt Lake City, Pres.; Mr. Mac F. Cahal, 406 W. 34th St., Kansas City 2, Executive Secy.

American Academy of Obstetrics and Gynecology, Dr. William F. Mengert, Dallas, Pres.; Dr. C. Paul Hodgkinson, Detroit, Secy.

American Academy of Ohthalmology and Otolaryngology. Dr. Frederick C. Cordes, San Francisco. Pres.; Dr. W. L. Benedict, 100 First Ave. Bldg., Rochester, Minn., Secy.

American Academy of Pediatrics, Chicago, Oct. 4-7, 1954. Dr. Roger L. J. Kennedy, Rochester, Minn., Pres.; Dr. E. H. Christopherson, 610 Church St., Evanston, Ill., Secy.

American Association for Thoracic Surgery, Montreal, Canada, May 3-5, 1954. Dr. Emile F. Holman, San Francisco, Pres.; Dr. Paul C. Samson, 2938 McClure St., Oakland 9, Calif., Secy.

American Association of Genito-Urinary Surgeons, Shawnee-on Delaware, Pa., May 26-28, 1954. Dr. Vincent J. O'Conor, Chicago, Pres.; Dr. Norris J. Heckel, 122 S. Michigan Ave., Chicago 3, Secy.

American Association of Obstetricians, Gynecologists and Abdominal Surgeons, Hot Springs, Va., Sept. 9-11, 1954. Dr. Herbert F. Schmitz, Chicago, Pres.; Dr. F. R. Lock, Bowman Gray School, Winston-Salem, N. C., Secy.

American Cancer Society. Dr. Guy Aud, Louisville, Ky., Pres.; Mr. M. R. Runyon, 47 Beaver St., New York, Executive Vice-Pres.

American College of Allergists, Miami Beach, April 5-10, 1954. Dr. M. Murray Peshkin, New York, Pres.; Dr. Fred W. Wittich, 423 La Salle Medical Bldg., Minneapolis 2, Secy.

American College of Chest Physicians, San Francisco, June 17-20, 1954. Dr. Alvis E. Greer, Houston, Pres.; Mr. Murray Kornfeld, 112 E. Chestnut St., Chicago 11, Executive Secy.

American College of Physicians, Chicago, April 5-9, 1954. Dr. Leroy H. Sloan, Chicago, Pres.; Mr. E. R. Loveland, 4200 Pine St., Philadelphia 4, Secy.

American College of Radiology, Chicago, Feb. 5, 1954. Dr. John D. Camp, Los Angeles, Pres.; Mr. W. C. Stronach, 20 N. Wacker Drive, Chicago 6, Executive Secy.

American College of Surgeons, Atlantic City, N. J., Nov. 14-18, 1954. Dr. Fred W. Rankin, Lexington, Ky., Pres.; Dr. Michael L. Mason, 40 E. Erie St., Chicago 11, Secy.

American Congress of Physical Medicine and Rehabilitation, Washington, D. C., Sept. 6-11, 1954. Dr. William B. Snow, New York, Pres.; Dr. Frances Baker, 1 Tilton Ave., San Mateo, Calif., Secy.

American Dermatological Association, White Sulphur Springs, W. Va., April 13-17, 1954. Dr. Louis A. Brunsting, Rochester, Minn., Pres.; Dr. J. Lamar Callaway, Duke Hospital, Durham, N. C., Secy.

American Gastro-Enterological Association, San Francisco, June 18-19, 1954. Dr. J. M. Ruffin, Durham, N. C., Pres.; Dr. H. Marvin Pollard, 1313 E. Ann St., Ann Arbor, Mich., Secy.

American Gynecological Society, Hot Springs, Va., May 20-22, 1954. Dr. Richard W. TeLinde, Baltimore, Pres.; Dr. John I. Brewer, 104 S. Michigan Ave., Chicago, Secy.

American Heart Association, Chicago, April 1-4, 1954. Dr. Robert L. King, Seattle, Pres.; Dr. William H. Bunn, 44 E. 23d St., New York 10, Secy.

American Hospital Association. Dr. Edwin L. Crosby, Chicago, Pres.; Mr. George P. Bugbee, 18 E. Division St., Chicago, Executive Secy.

American Laryngological, Rhinological, and Otological Society, Boston, May 25-27, 1954. Dr. LeRoy A. Schall, Boston, Pres.; Dr. C. S. Nash, 277 Alexander St., Rochester 7, N. Y., Secy.

American Neurological Association, Atlantic City, N. J., June 14-16, 1954. Dr. Roland P. Mackay, Chicago, Pres.; Dr. H. Houston Merritt, 710 W. 168th St., New York 32, Secy.

American Ophthalmological Society, Glacier Park, Mont., June 16-18, 1954. Dr. William L. Benedict, Rochester, Minn., Pres.; Dr. M. C. Wheeler, 30 W. 59th St., New York 19, Secy.

American Orthopedic Association, Bretton Woods, N. H., June 6-9, 1954. Dr. A. R. Shands, Jr., Wilmington, Del., Pres.; Dr. George O. Eaton, 4 E. Madison St., Baltimore 2, Secy.

American Pediatric Society, Buck Hill Falls, Pa., May 3-5, 1954. Dr. Rustin McIntosh, New York, Pres.; Dr. Aims McGuinness, 237 Medical Laboratory, University of Pennsylvania, Philadelphia 46, Secy.

American Proctologic Society, Los Angeles, June 2-5, 1954. Dr. Wendell Green, Toledo, Ohio, Pres.; Dr. Stuart T. Ross, 131 Fulton Ave., Hempstead, N. Y., Secy.

American Psychiatric Association, St. Louis, May 3-7, 1954. Dr. Kenneth E. Appel, Philadelphia, Pres.; Dr. R. Finley Gayle, 6300 Three Chopt Rd., Richmond 21, Va., Secy.

American Public Health Association, Buffalo, N. Y., Oct. 11-15, 1954. Dr. Wilton L. Halverson, San Francisco, Pres.; Dr. R. W. Atwater, 1790 Broadway, New York 19, Executive Secy.

American Society of Anesthesiologists. Dr. Ralph T. Knight, Minneapolis, Pres.; Dr. J. E. Remlinger, Jr., 188 W. Randolph St., Chicago, Secy.

American Society of Clinical Pathologists, Washington, D. C., Sept. 6, 1954. Dr. John R. Schencken, Omaha, Pres.; Dr. Clyde G. Culbertson, Indiana University School of Medicine, Indianapolis, Secy.

American Surgical Association, Cleveland, April, 1954. Dr. Howard C. Naffziger, San Francisco, Pres.; Dr. R. Kennedy Gilchrist, 59 East Madison St., Chicago 3, Secy.

American Urological Association, New York, May 31-June 3, 1954. Dr. J. A. Campbell Colston, Baltimore, Md., Pres.; Dr. C. H. deT. Shivers, 121 S. Illinois Ave., Atlantic City, N. J., Secy.

Association of American Physicians and Surgeons, Jackson, Miss., April 1-3, 1954. Dr. Thomas G. Goldsmith, Greenville, S. C., Pres.; Mr. Harry E. Northam, 360 N. Michigan Ave., Chicago 1, Executive Secy.

International College of Surgeons, U. S. Chapter, Chicago, Sept. 7-10, 1954. Dr. William R. Lovelace, Albuquerque, N. M., Pres.; Dr. Karl Meyer, 1516 Lake Shore Drive, Chicago, Secy.

National Tuberculosis Association, Atlantic City, N. J., May 17-21, 1954. Mr. Mark H. Harrington, Denver, Pres.; Mr. Kemp D. Battle, 1790 Broadway, New York 19, Secy.

Radiological Society of North America. Dr. Ira H. Lockwood, Kansas City, Mo., Pres.; Dr. D. S. Childs, 713 E. Genesee, Syracuse 2, N. Y., Secy.

Southern Medical Association, St. Louis, 1954. Dr. Alphonse McMahon, St. Louis, Pres.; Mr. C. P. Loranz, 1020 Empire Bldg., Birmingham 3, Ala., Secy.

Southern Psychiatric Association, Louisville, October, 1954. Dr. John D. Trawick, Louisville, Ky., Pres.; Dr. Joseph L. Knapp, 210 N. Westmoreland, Dallas, Secy.

Southern Surgical Association. Dr. I. A. Bigger, Richmond, Va., Pres.; Dr. John C. Burch, 2112 West End Ave., Nashville 5, Tenn., Secy.

Southwest Allergy Forum, New Orleans, May 9-11, 1954. Dr. Henry D. Ogden, New Orleans, Pres.; Dr. Stanley Cohen, 1441 Delachaise St., New Orleans, Secy.

Southwest Regional Cancer Conference. Secy., 209 Medical Arts Bldg., Fort Worth.

Southwestern Medical Association, El Paso, 1954. Dr. Willard W. Schuessler, El Paso, Pres.; Dr. Celso Stapp, 800 Montana, El Paso, Secy.

Southwestern Surgical Congress, Oklahoma City, Sept. 27-29, 1954. Dr. Philip B. Price, Salt Lake City, Pres.; Dr. C. R. Rountree, 1227 Classen, Oklahoma City 3, Secy.

Tri-State Medical Assembly, Fall, 1954. Dr. Joe D. Nichols, Atlanta, Pres.; Dr. Broox C. Garrett, 940 Margaret Pl., Shreveport, La., Secy.

United States-Mexico Border Public Health Association, Albuquerque, N. M., April, 1954. Mr. Richard F. Poston, Dallas, Pres.; Dr. Sidney B. Clark, 314 U. S. Court House, El Paso, Secy.

STATE

Private Clinics and Hospitals Association of Texas. Dr. W. R. Swanson, Taylor, Pres.; Dr. John Dupree, Levelland, Secy.

Texas Academy of General Practice, Galveston, Sept. 9-11, 1954. Dr. C. Forrest Jorns, Houston, Pres.; Dr. Woodson W. Harris, 1410 Nickerson, Austin, Secy.

Texas Academy of Internal Medicine. Dr. Martin S. Buehler, Dallas, Pres.; Dr. George M. Jones, 1314 Medical Arts Bldg., Dallas, Secy. Meetings restricted to members.

Texas Air-Medics Association, San Antonio, May 2-3, 1954. Dr. George L. Gallaher, Harlingen, Pres.; Dr. J. S. Minnett, 2512 Welborn, Dallas, Secy.

Texas Association of Blood Banks, Abilene, Dec. 3-4, 1954. Dr. C. T. Ashworth, Fort Worth, Pres.; Miss Marjorie Saunders, 3500 Gaston Ave., Dallas, Secy.

Texas Association of Obstetricians and Gynecologists, Waco, Feb. 19-20, 1954. Dr. D. D. Wall, San Angelo, Pres.; Dr. Carey Hiett, 815 Fifth Ave., Fort Worth, Secy.

Texas Chapter, American College of Chest Physicians, San Antonio, May 2, 1954. Dr. Henry H. Hoskins, San Antonio, Pres.; Dr. Walter C. Brown, 1733 S. Brownlee Blvd., Corpus Christi, Secy.

Texas Club of Internists. Dr. Joseph F. McVeigh, Fort Worth, Pres.; Dr. Charles Darnall, Capital National Bank Bldg., Austin, Secy.

Texas Dermatological Society, San Antonio, May 2, 1954. Dr. Maurice Barnes, Waco, Pres.; Dr. Thomas L. Shields, 1216 Pennsylvania Ave., Fort Worth, Secy.

Texas Diabetes Association, San Antonio, May 2, 1954. Dr. Raymond L. Gregory, Galveston, Pres.; Dr. Edmond K. Doak, 501 Hermann Professional Bldg., Houston, Secy.

Texas Division, American Cancer Society, Austin, Jan. 14-15, 1954. Mr. Travis Wallace, Dallas, Pres.; Mr. J. Louis Neff, 1609 Colorado, Austin, Executive Director.

Texas Heart Association, San Antonio, May 3, 1954. Dr. Joseph McVeigh, Fort Worth, Pres.; Miss Roberta Miller, 412 Fidelity Union Life Bldg., 1511 Bryan St., Dallas, Executive Secy.

Texas Hospital Association, Houston, May 18-20, 1954. Mr. W. U. Paul, El Paso, Pres.; Mrs. Ruth Barnhart, 2210 Main St., Dallas, Secy.

Texas Neuropsychiatric Association, San Antonio, May 2, 1954. Dr. James Blair, San Antonio, Pres.; Dr. John L. Otto, John Sealy Hospital, Galveston, Secy.

Texas Orthopedic Association, San Antonio, May 3, 1954. Dr. Edward T. Smith, Houston, Pres.; Dr. Margaret Watkins, 3629 Fairmount St., Dallas, Secy.

Texas Pediatric Society, Fort Worth, October, 1954. Dr. Robert L. Moore, Dallas, Pres.; Dr. James N. Walker, 3616 Tulsa Way, Fort Worth, Secy.

Texas Public Health Association, Galveston, Feb. 14-17, 1954. Dr. Roy G. Reed, La Marque, Pres.; Mr. Earle W. Sudderth, Dallas County Health Department, Court House, Dallas, Executive Secy.

Texas Radiological Society, Dallas, Jan. 29-30, 1954. Dr. J. E. Miller, Dallas, Pres.; Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth, Secy.

Texas Railway and Traumatic Surgical Association, San Antonio, May 3, 1954. Dr. William E. Crump, Wichita Falls, Pres.; Dr. W. D. Marrs, 306 Broadway, Fort Worth, Secy.

Texas Rheumatism Association. Dr. Alfred H. Harris, Dallas, Pres.; Dr. Charles H. Cornwell, Marlin, Secy.

Texas Society for Mental Health, Waco, March 4-6, 1954. Mrs. Frank Schoonover, Fort Worth, Pres.; Mrs. Elizabeth F. Gardner, 2504 Jarratt Ave., Austin 21, Executive Secy.

Texas Society of Anesthesiologists, San Antonio, May 2, 1954. Dr. James B. Robinett, Jr., Houston, Pres.; Dr. C. R. Allen, John Sealy Hospital, Galveston, Secy.

Texas Society of Gastroenterologists and Proctologists, San Antonio, May 3, 1954. Dr. Cecil O. Patterson, Dallas, Pres.; Dr. W. T. Arnold, 1402 Hermann Prof. Bldg., Houston, Secy.

Texas Society of Ophthalmology and Otolaryngology. Dr. Dan Brannin, Dallas, Pres.; Dr. Lyle Hooker, 627 Esperson Bldg., Houston, Secy.

Texas Society of Pathologists, Galveston, Jan. 31, 1954. Dr. A. O. Severance, San Antonio, Pres.; Dr. Lloyd R. Hershberger, Shannon Memorial Hospital, San Angelo, Secy.

Texas Surgical Society, Waco, April 5-6, 1954. Dr. Dudley Jackson, Sr., San Antonio, Pres.; Dr. Albert W. Hartman, 414 Navarro St., San Antonio 5, Secy.

Texas Tuberculosis Association, Dallas, April 9-10, 1954. Dr. R. G. McCorkle, San Antonio, Pres.; Miss Pansy Nichols, 208 E. Ninth, Austin, Executive Secy.

Texas Urological Society, Fort Worth, Feb. 22, 1954. Dr. J. Reese Blundell, Houston, Pres.; Dr. Dolphus E. Compere, 1415 Pennsylvania Ave., Fort Worth, Secy.

DISTRICT

First District Society, Pecos, Feb. 12, 1954. Dr. John W. O'Donnell, Alpine, Pres.; Dr. H. D. Garrett, First National Bldg., El Paso, Secy.

Second District Society, Sweetwater, April 15, 1954. Dr. T. D. Young, Sweetwater, Pres.; Dr. Francis Hood, Sweetwater, Secy.

Third District Society, Amarillo, April 13, 1954. Dr. Robert A. Neblett, Canyon, Pres.; Dr. James T. Hall, 1626 Fifteenth St., Lubbock, Secy.

Fourth District Society, San Angelo, Fall, 1954. Dr. P. M. Wheelis, Brownwood, Pres.; Dr. James N. White, San Angelo, Secy.

Fifth and Sixth Districts Society, Corpus Christi, July, 1954. Dr. Y. C. Smith, Sr., Corpus Christi, Pres.; Dr. Robert J. Sigler, 1126 Third Street, Corpus Christi, Secy.

Seventh District Society, Austin, Feb. 18, 1954. Dr. Benjamin Clary Bates, Austin, Pres.; Dr. William McLean, 4618 Burnet Road, Austin, Secy.

Eighth District Society. Dr. Howard Z. Fretz, Wharton, Pres.; Dr. George E. Glover, Jr., Victoria, Secy.

Ninth District Society, Brenham, March 25, 1954. Dr. George D. Bruce, Baytown, Pres.; Dr. Lyman C. Blair, 1212 Rothwell, Houston, Secy.

Tenth District Medical Society. Dr. Edmund D. Jones, Beaumont, Pres.; Dr. O. W. Harris, Jr., Liberty, Secy.

Eleventh District Society, Crockett, Spring, 1954. Dr. John Dean, Crockett, Pres.; Dr. Marlin T. Braswell, Henderson, Secy.

Twelfth District Society, Waco, Jan. 12, 1954. Dr. Howard Dudgeon, Jr., Waco, Pres.; Dr. Walter B. King, Jr., Waco, Secy.

Thirteenth District Society, Fort Worth, 1954. Dr. William E. Crump, Wichita Falls, Pres.; Dr. Robert D. Moreton, 815 Medical Arts Bldg., Fort Worth, Secy.

Fourteenth District Society, Denton, 1954. Dr. J. W. Atchison, Gainesville, Pres.; Dr. L. W. Johnston, 502 W. College St., Terrell, Secy.

Fifteenth District Society, Longview, April 20, 1954. Dr. L. E. Rutledge, Daingerfield, Pres.; Dr. George Tate, Longview, Secy.

CLINICS

Dallas Southern Clinical Society, Dallas, March 15-18, 1954. Miss Helga Boyd, Medical Arts Bldg., Dallas 1, Executive Secy.

Central Texas Spring Clinic, Waco, April 7, 1954. Dr. Walter B. King, 2320 Columbus Ave., Waco, Secy.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 26-28, 1954. Dr. John M. Smith, Jr., 205 Camden St., San Antonio, Secy.

New Orleans Graduate Medical Assembly, New Orleans, March 8-11, 1954. Dr. Maurice E. St. Martin, Room 103, 1430 Tulane Ave., New Orleans 12, Secy.

North Texas-Southern Oklahoma Fall Clinical Conference. Dr. C. H. Wilson, Wichita Falls, Chairman.

Oklahoma City Clinical Society Conference, Oklahoma City, Oct. 25-28, 1954. Miss Alma F. O'Donnell, 512 Medical Arts Bldg., Oklahoma City 2, Executive Secy.

Postgraduate Medical Assembly of South Texas, July 19-21, 1954. Dr. Edward T. Smith, 906 Hermann Professional Bldg., Houston, Secy.

State Tumor Conference, Wichita Falls, March 31, 1954. Dr. Bailey R. Collins, 925½ Scott Street, Wichita Falls, Director.

BOARD EXAMINATIONS

Texas State Board of Examiners in Basic Sciences. Mrs. Betty Ratcliff, 407 Perry-Brooks Bldg., Austin, Chief Clerk.

Texas State Board of Medical Examiners, Fort Worth, June 21-23, 1954. Dr. M. H. Crabb, 1714 Medical Arts Bldg., Fort Worth, Secy.

American Academy of Allergy

Texas physicians are invited to attend the annual meeting of the American Academy of Allergy to be held in Houston on February 1, 2, and 3. No charge will be made for registration, although those who wish to attend the postgraduate courses in clinical allergy, advanced subjects (seminar), and allergenic molds and pollens, scheduled for January 29-31, will pay $40, $15, and $35 respectively.

Specialists from throughout the United States and representatives from several foreign countries will present papers, and scientific and commercial exhibits will be displayed. A program for visiting ladies also is planned.

The Committee on Local Arrangements includes Drs. Homer E. Prince, chairman; Ralph Bowen, DeWitt H. Hotchkiss, Jr., Richard L. Etter, Jack M. Rose, and Warren F. Kahle. The Hostess Committee is headed by Mrs. Homer E. Prince, chairman.

Hotel reservations should be made with the Shamrock Hotel, Houston, and registration for the postgraduate courses with the American Academy of Allergy, 208 E. Wisconsin Avenue, Milwaukee 2. No advance registration is necessary for attending the meeting.

LEGISLATIVE PREVIEW

As the second session of the Eighty-Third Congress gets under way, the Washington office of the American Medical Association has outlined some of the pending legislation of interest to the medical profession.

Social Security.—Awaiting action is the administration's plan to extend social security coverage to more than 10,000,000 additional persons, including physicians. The American Medical Association, as well as other groups of the self-employed, has opposed this type of legislation consistently. Instead, the AMA recommends the passage of a measure to allow self-employed persons to defer income tax payments on a portion of their incomes, placed in restricted pension funds and obtainable in the form of benefits only in case of disability or at the specified retirement age. Such provisions were included in bills first introduced as Reed-Keogh bills and now identified as Jenkins-Keogh because of the sponsors.

Other possible amendments to the social security law involve total and permanent disability payments and waiver of old age and survivor insurance premiums for the disabled so that their final pensions will not be reduced because of periods when they had little or no income. In each of these, medical determinations would be required, threatening an expansion of the federal medical program, placing the physicians in an unreasonable role, or calling for compulsory rehabilitation. The AMA is not opposed to the objective of the proposals but suggests that pension rates be based on the ten best earning years instead of that premiums be waived, thus obviating the need for medical determinations.

It is expected that Congress again will be asked to pass legislation providing free hospitalization under the old age and survivors insurance laws for all persons past the age of 65 years and covered by the insurance.

Military Personnel and Veterans.—In line with the recent action of the Veterans Administration in amending its forms to require more financial information from veterans applying for hospitalization of nonservice connected disabilities, who must state that they cannot afford private care, Congress may want further clarification of the government's obligations to veterans. It is thought that special effort will be made to expand medical benefits to veterans by such methods as increasing the periods in which certain diseases may be presumed to be of service origin. The position of the American Medical Association with respect to medical care of veterans has been declared as (1) for the best possible care by the Veterans Administration of all veterans with service connected conditions, (2) for Veterans Administration care of long-term tuberculous and neurologic patients if the veterans cannot pay for it themselves and until local and state facilities are adequate, and (3) against Veterans Administration care of all other cases of nonservice connected conditions, these being the responsibility of the veteran, his family, or his community.

Efforts to implement the Moulton Commission's recommendations for broadening the medical care program for military dependents has been promised by the Defense Department. The commission favors caring for as many dependents as possible at military installations, with others receiving private care primarily at the expense of the federal government. The American Medical Association in December proposed that in the United States military personnel and facilities be used for dependents only if private facilities are inadequate.

Another measure which the Defense Department is recommending would provide government scholarships for medical, dental, and nursing study with the recipients obligated for government service at the rate of one year for every year of the scholarship. The AMA House of Delegates referred to the Board of Trustees the problem of whether or not such legislation should be endorsed.

Health Insurance.—Bills probably will be offered to provide government subsidy of private health insurance plans.

Bricker Amendment.—Holding over from the previous session is the Bricker resolution to check the treaty-making powers of the President. The AMA has endorsed such a measure.

Relationship of Governments.—Two commissions appointed last year, one headed by Clarence Manion and the other by former President Herbert Hoover, are studying the relationships between the federal government on one hand and state and local governments on the other and also the operations of the executive branch of the federal government. The Manion Commission is supposed to have a report ready by March but may ask for more time; the Hoover Commission has until next year to report.

HEALTH INSURANCE INCREASE

Accident and health coverage in the United States increased to a new record high level in 1952, according to the Health Insurance Council's annual survey released in September, 1953.

In Texas, 3,800,000 persons were protected against the expense of hospitalization at the end of 1952, 3,697,000 against surgical expenses, and 1,743,000 against medical expense.

More than $2 billions cash benefits were received in 1952 in the nation, with about half this amount to cover the cost of hospitalization. The total number of persons with hospitalization insurance increased 7 per cent over 1951. There was a 12 per cent increase in the number of persons protected against the cost of operations under surgical expense coverage, and the number of those protected against doctors' bills under medical expense coverage shows an increase of 29 per cent over 1951.

The council stated that at the close of 1952 more than 38 million persons were protected against the loss of income due to disability, and nearly 700,000 had major medical expense coverage, the newest form of voluntary health protection designed to help meet the costs of very serious illness.

Organizations covered in the council report include insurance companies, Blue Cross, Blue Shield, and various other independent plans sponsored by business and industry, employee benefit associations, and private group clinics.

COX RESIGNS AS STATE HEALTH OFFICER

Dr. George W. Cox, State Health Officer of Texas for the past seventeen years, offered his resignation, effective March 1, when the State Board of Health met in Austin on December 14. The board accepted his resignation with the request that he remain in office until a successor can be selected and pointed out that during his term the services of the State Department of Health have been extended to "every county and municipality in Texas with a degree of efficiency equal to the best public health services in the nation."

AMERICAN GERIATRICS SOCIETY

The American Geriatrics Society will meet June 17-19 in San Francisco, immediately preceding the meeting of the American Medical Association. Physicians and other scientists interested in geriatrics may attend. Hotel reservations should be made through the San Francisco Convention and Visitors Bureau, 200 Civic Auditorium, San Francisco 2. Additional information may be obtained from the chairman of local arrangements, Dr. Laurance W. Kinsell, Highland Alameda County Hospital, 2701 Fourteenth Avenue, Oakland 6.

BLOOD BANKS FORM CLEARING HOUSE

The Texas Association of Blood Banks, meeting December 4 and 5 in Fort Worth, agreed to establish a clearing house for the exchange of blood between banks in the state. The clearing house will maintain an inventory of the types and quantity of blood available in participating banks, initiate the exchange of blood, record transactions and arrange for cancellation of indebtedness between banks, collect and pay processing fees, and maintain itself by a proration among member banks.

More than 100 delegates were present for the meeting, which included papers, panel and round-table discussions, a luncheon and a banquet, and a business meeting. Out-of-state participants in the program were Dr. John Upton, chairman of the California Blood Bank Commission, San Francisco; Mrs. Charles Hemphill, managing director of the Irwin Memorial Blood Bank, San Francisco; Dr. Sam Gibson, associate director of the blood program of the American National Red Cross, Washington; and Dr. Joseph A. Hertell, assistant national director of the American National Red Cross, Washington.

Dr. C. T. Ashworth, Fort Worth, was named president for the coming year; Dr. Jarrett Williams, Abilene, president-elect; Dr. O. J. Wollenman, Jr., Fort Worth, vice-president; Miss L. Jean Stubbins, M. T., Galveston, treasurer; and Miss Marjorie Saunders, Dallas, secretary. The 1954 meeting will be held December 3 and 4 in Abilene.

TEXAS RHEUMATISM ASSOCIATION

The Texas Rheumatism Association held its sixth annual meeting December 4 in Houston with Dr. Edward W. Boland, president-elect of the American Rheumatism Association, as special guest. Dr. J. C. Crager, Beaumont, president, presided; Dr. Jesse W. Hofer, Houston, was program chairman.

Dr. Alfred H. Harris, Dallas, was elected president; Dr. Howard C. Coggeshall, Dallas, first vice-president; Dr. Frank F. Parrish, Houston, second vice-president; and Dr. Charles H. Cornwell, Marlin, secretary-treasurer. Drs. C. C. Smith, Waco, and W. W. Moorman, Fort Worth, are new members of the board of governors.

The following program was presented:

Roentgenological Findings of Rheumatoid Arthritis and Rheumatoid Spondylitis—Dr. Vincent P. Collins, Houston.
Rheumatic Manifestations of Endocrine Diseases—Dr. E. L. Crain, Houston.
Mechanism of Action and Clinical Use of Probenecid in Gout—Dr. Ralph V. Ford, Houston.
Unusual Bone Tumors—Dr. John R. Thomas, Houston.
Conditions Producing Pain in Shoulder Girdle—Dr. Frank F. Parrish, Houston.
Neurological Disorders Simulating Rheumatic Diseases—Dr. Moses Ashkenazy, Houston.
Orthopedic Principles in Management of Rheumatoid Arthritis—Dr. Bedford F. Boylston, Houston.
Physical Medicine in Treatment of Rheumatoid Arthritis—Dr. Lewis A. Leavitt, Houston.
Present Status of Steroid Therapy in Rheumatoid Arthritis—Dr. Boland.

Dr. Boland also led informal discussions of the papers and an open question period. All other speakers are on the staff of Baylor University College of Medicine. Approximately eighty-five Texas physicians attended.

Reciprocity Applications Coming Due

Reciprocity applications must be complete thirty days prior to the time for examinations for licensure by the Texas State Board of Medical Examiners, Dr. M. H. Crabb, secretary, has reminded. The next examination session will be held June 21-23 at the Texas Hotel in Fort Worth.

Texans Enter College of Surgeons

Sixty-three Texas doctors of medicine became fellows in the American College of Surgeons at its 1953 convocation in October. The names released by the college are as follows:

Mack F. Bowyer, Abilene; William R. Klingensmith, Jr. and Walter C. Watkins, Amarillo; Percy R. Fayle and Robert O. Whitson, Baytown; Lamar C. Bevil, Kenneth T. Miller, and Charles L. Pentecost, Beaumont; Houston J. Zinn, Big Spring; George Dudley Moore, Jr. and Charles R. Nester, Canyon; Jack F. Fox, Childress; Sam R. Burnett, J. Harold Cheek, Vanda A. Davidson, Jr., Foster Fuqua, W. N. Fuqua, Jr., Arthur P. Gottlich, Henry G. Montgomery, Philip D. Newell, Donald L. Paulson, Robert R. Shaw, and Nathan Tobolowsky, Dallas; Haskell D. Hatfield and Morton H. Leonard, El Paso; J. Robert Harris, Rex J. Howard, and Hub E. Isaacks, Fort Worth.

Also Clifford C. Snyder, Galveston; George W. Duncan, Harlingen; Moses Ashkenazy, Frederick H. Aves, Bedford F. Boylston, Oscar Creech, Jr., Loren W. Haus, Boyd W. Haynes, Jr., George Robert Kolodny, Milbourne K. Leggett, Jack H. Mayfield, John W. Overstreet, George E. Rohrer, Bryan V. Williams, and Edward Carson Williams, Houston; J. Collier Rucker, Jacksonville; James H. Johnson, Jr., Longview; Robert T. Canon and Archie Lee Hewitt, Lubbock; Milton J. Loring and J. S. Roden, Midland; John B. Case, Graham M. Coffman, Phillip L. Day, Paul L. Gorsuch, James E. Pridgen, William W. Sawtelle, and Edwin M. Sykes, Jr., San Antonio; Hanes H. Brindley, Elihu I. Klein, and John F. McKenney, Jr., Temple; H. Gates Brelsford and Madison J. Lee, Jr., Tyler; Thomas R. Hunter, Jr., Wellington; and Charles H. Wilson, Wichita Falls.

Neuropsychiatric Session in Little Rock

Interested professional personnel have been invited to attend the sixth annual neuropsychiatric meeting to be held at the Veterans Administration Hospital, North Little Rock, February 25 and 26. There will be no charge for registration.

Guest lecturers who will participate include Drs. Kenneth E. Appel, president, American Psychiatric Association, Philadelphia; Franz Alexander, director, Chicago Institute for Psychoanalysis, Chicago; Francis J. Braceland, psychiatrist-in-chief, Institute of Living, Hartford, Conn.; Richard L. Jenkins, chief, Research Section, Psychiatry and Neurology Service, Department of Medicine and Surgery, Veterans Administration, Washington; William Malamud, professor of psychiatry, Boston University School of Medicine, Boston; Jules H. Masserman, neuropsychiatrist, Chicago; Henry G. Schwartz, professor of neurological surgery, Washington University, St. Louis; Harvey J. Tompkins, director, Psychiatry and Neurology Service, Department of Medicine and Surgery, Veterans Administration, Washington; Gregory Zilboorg, psychoanalyst, New York.

In addition to scientific sessions, there will be exhibits prepared by hospital staff members and a dinner the evening of February 25 at which Dr. Appel will be the principal speaker.

Additional information may be obtained from Dr. Ewin S. Chappell, director of professional education at the hospital.

EL PASO HAS NEW MEDICAL CENTER

A new El Paso medical center has nine professional buildings containing thirty-five suites and offices for doctors and dentists. A tenth building will house a prescription pharmacy and a restaurant.

The center is centrally located near the hospitals and will have ample parking facilities.

New Orleans Graduate Medical Assembly

The New Orleans Graduate Medical Assembly will hold its seventeenth annual meeting March 8-11, to be followed by a postclinical tour to Hawaii from Los Angeles by airplane and ship, March 14-April 6. Eighteen guest speakers will participate in the scientific program. Also on the agenda are symposiums, clinicopathologic conferences, round-table luncheons, three-dimensional surgical motion pictures, medical motion pictures, and technical exhibits.

The speakers will be Drs. Perry P. Volpitto, Augusta, Ga.; Earl D. Osborne, Buffalo; Julian M. Ruffin, Durham, N. C.; Allan C. Barnes, Cleveland; Walter C. Alvarez, St. Paul; William D. Stroud, Philadelphia; Lawrence C. Kolb, Rochester, Minn.; Nicholson J. Eastman, Baltimore; A. D. Ruedemann, Detroit; Oscar L. Miller, Charlotte, N. C.; Francis L. Lederer, Chicago; Emmerich von Hamm, Columbus, Ohio; Philip M. Stimson, New York; Brian Blades, Washington, D. C.; Samuel F. Marshall, Boston; Orvar Swenson, Boston; Charles D. Creevy, Minneapolis; and one other to be announced.

Registration fee of $20 is all-inclusive. Information concerning the assembly meeting or the tour is available from the secretary, Dr. Maurice E. St. Martin, 1430 Tulane Avenue, Room 103, New Orleans 12.

HYPERTENSION MORTALITY

Several tentative conclusions concerning mortality from essential hypertension have been reached as the result of a ten-year follow-up study of 646 persons suffering from the affliction by Drs. Robert S. Palmer and Hugo Meunch, Boston. Mortality was found to be greatest among young people, decreasing rapidly with age, and definitely lower in women, according to the doctors' report in the September 5 issue of *The Journal of the American Medical Association.* At the time the report was prepared, 453 patients were alive. The conclusions follow:

1. Relative risk is highest in the young and decreases rapidly with age.

2. Mortality in women is definitely lower (except in the more severe types) and increase of severity is slower. In the higher ages with the milder forms of hypertension, the mortality of women is approximately that of the general population.

3. The average age increases from those with very mild hypertension to those with more severe types. Each of the more severe types must be to some extent a later development of the previous one.

4. Duration of the disease, apart from advancing age, does not seem to have any effect on mortality, except in the most severe type in which short duration is associated with higher mortality at youngest ages.

5. With each increasing degree of hypertension, patients die at a faster rate than that expected of the general public. In general, this increase in mortality tends to diminish with time under observation or higher ages.

Treatment of the patients observed by the physicians was chiefly dietary according to conventional indications; ordinary rules of good hygiene and simple psychotherapy were varied and adapted to the particular patient.

Chest Disease Course Set for New Orleans

Postgraduate courses in diseases of the chest are being sponsored by the American College of Chest Physicians in New Orleans, February 15-19, and in Philadelphia, March 15-19. Tuition for each course is $75. Additional details may be obtained from the Executive Director, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11.

OKLAHOMA ACADEMY OF GENERAL PRACTICE

The Oklahoma Academy of General Practice will meet in Tulsa February 15-17 to hear the following speakers: Dr. William F. Guerriero, Dallas; Dr. Horace L. Hodes, New York; Dr. Manuel E. Lichtenstein, Chicago; Dr. Perrin H. Long, Brooklyn; Dr. Carlo Scuderi, Chicago; Dr. Harry Wilkins, Oklahoma City; and other outstanding speakers presented by Lederle Laboratories in conjunction with the convention. There is no registration fee, and Texas physicians will be welcome.

A public forum on children's diseases will be held February 16, and a banquet, breakfast, and entertainment also are scheduled. A luncheon and style show will be held for wives.

Further information may be obtained from the president, Dr. Marshall O. Hart, 1228 South Boulder, Tulsa 5.

Aspirin in Respiratory Disorders

In patients suffering from such acute respiratory infections as influenza, bronchitis, and the common cold, aspirin produces normal temperatures more speedily than Terramycin and Erythromycin, according to an article in the September 26 *Journal of the American Medical Association.* The findings of a team of United States Air Force doctors show that aspirin brought the patients' temperature down to normal in an average of thirty hours as compared with forty-one hours for Terramycin and forty-two hours for Erythromycin. The evidence indicates that the antibiotic drugs had no influence on the course of these diseases nor was the use of these drugs necessary since no secondary bacterial infections developed in any of the patients tested.

"The patients who were given aspirin usually felt better sooner than those who were receiving either terramycin or erythromycin," the article states. The patients in the study were 150 airmen with respiratory diseases, the majority believed to be of the influenza-like type. A 600 mg. dose of aspirin was administered to a group of 54 patients with follow-up doses for each group at six-hour intervals. With the exception of some tiredness and a dry cough, such symptoms as headache, general muscular aches, and sore throats disappeared once the fever in all patients returned to normal.

Since no specific cure for common virus infections is now available, the doctors state, "any measure that will limit or minimize the duration of the illness is important."

The study was conducted by Capt. Philip N. Jones, Major Roy S. Bigham, Jr., and Lt. Phil R. Manning, from the Chanute Air Force Base in Illinois.

Medical Aid Needed in Asia

The barest necessities of hospital and medical equipment are needed in the nearly 300 hospitals in India and surrounding Southeast Asian countries, and a joint program of CARE and the World Health Organization, approved by the American Medical Association and its Auxiliary, is attempting to fill this need. About $1,750,000 is needed to take care of the minimum requirements of bed linen, surgical instruments, sterilizers, and laboratory apparatus, according to a survey by WHO.

Purchasing, shipping, and delivering has been put in the hands of CARE, a nonprofit, cooperative agency composed of twenty-seven major American welfare agencies which for the past seven years has channelled American aid in the form of 15,000,000 food, textile, and "self-help" tool packages, books, and other equipment totalling more than $150,000,000 to needy people and institutions in more than thirty overseas countries.

Donations to the project may be forwarded to CARE, 660 First Avenue, New York 16.

PERSONALS

Dr. Lawrence M. Shefts, San Antonio, lectured and performed surgery for a postgraduate course in chest surgery at the medical school of the University of Nuevo Leon, Monterrey, Mexico, in October.

Dr. G. V. Brindley, Temple, has been elected chairman of the Medical and Scientific Committee of the American Cancer Society. Dr. Brindley is chairman of the executive committee of the Texas division and a member of the national board of directors.

Dr. Oren H. Chandler, Ballinger, has been named president of the board of trustees of Howard Payne College, Brownwood. Dr. Chandler has been a trustee for twelve years and vice-president the past two years.

Dr. W. Earl Reed, San Juan, is president of the Pharr Kiwanis Club.

Dr. Perry R. Jeter, Childress, was named Lion of the Month for September by the local Lions Club.

Dr. J. R. McGee, New Boston, first General Practitioner of the Year in Texas, and *Dr. J. H. McCracken,* Mineral Wells, past president of the Texas Medical Association, were among the physicians in practice fifty years or more who were honored by the University of Tennessee, Memphis, this fall. Certificates of honor were presented to them in recognition of their service.

Dr. John Q. Rounsaville, Dallas, has been named medical director of the National Bankers Life Insurance Company.

The father of *Dr. Warren G. Knox,* Fort Worth, died November 23 at Roby.

Mrs. Felix Peebles, mother of *Dr. Felix Peebles, Jr.* of Lufkin, died in Jefferson October 21.

Dr. and Mrs. John R. Thomas, Corpus Christi, were honored at a reception given by their children October 24 in Corpus Christi in observance of their golden wedding anniversary.

Dr. Albert Wilson Harrison and Miss Florence Adelaide Gask were married October 31 in Daytona Beach, Fla. They reside in Galveston.

Dr. and Mrs. O. T. Kirksey, Galveston, and *Dr. and Mrs. Bernard Rosen,* Corsicana, are recent parents of sons; *Dr. and Mrs. R. V. Jones,* Houston, are parents of a daughter.

Dr. and Mrs. W. Compere Basom, El Paso, are parents of a boy, Robert Compere, born December 5.

Hospital Construction Progress

The Texas State Department of Health recently issued its annual plan for construction of hospitals and public health centers, which in reality is a report of progress made since funds from the Hill-Burton hospital survey and construction act adopted by the United States Congress became available in 1947 and a summary of anticipated activity making use of those funds in the future.

The report shows that 133 projects have been approved, of which 86 have been completed, 21 are under construction, 8 are not yet under contract, and 18 have been withdrawn. The 115 projects completed or now scheduled for construction include 89 general hospitals, 4 hospitals for chronically ill patients, 6 tuberculosis hospitals, 2 hospitals for nervous and mental diseases, 6 public health centers, 3 nurses' homes, 4 equipment projects, and 1 expansion of the state laboratory. The hospitals provide for an additional 8,988 beds.

For the future, the areas of greatest need and specific ameliorative projects are defined in the report. Possible sponsoring agencies for these projects will be encouraged to apply for assistance, but the proposed overall plan will be modified as necessary.

Operating under the general direction of the State Board of Health, the hospital survey and construction division of the State Department of Health also has a state advisory council of twelve members. Four vacancies on the council now exist; Drs. R. W. Kimbro, Cleburne; Joe D. Nichols, Atlanta; and James T. Lee, Wichita Falls, are among the members.

CARDIOVASCULAR COURSE IN HOUSTON

A combination of hospital ward rounds, conferences, and demonstrations and of evening lectures will be used in the cardiology course to be held February 15-19 in Houston. The course is sponsored jointly by the University of Texas Postgraduate School of Medicine and Baylor University College of Medicine in association with the Houston Heart Association, Texas Medical Association, Texas State Department of Health, Houston Society of Internal Medicine, and Houston Academy of General Practice.

Visiting lecturers will include Dr. Robert H. Bayley, professor of medicine, University of Oklahoma, Oklahoma City; Dr. Arthur Grollman, professor of experimental medicine, Southwestern Medical School of the University of Texas, Dallas; Dr. George Herrmann, professor of medicine, University of Texas Medical Branch, Galveston; Dr. Donald Seldin, professor of medicine, Southwestern Medical School, Dallas; and Dr. Henry M. Winans, emeritus professor of medicine, Southwestern Medical School, Dallas. Fifteen local faculty members will participate, and the staffs of Hermann, Jefferson Davis, M. D. Anderson, Memorial, Methodist, St. Joseph's, and Veterans Administration Hospitals will cooperate.

Newer diagnostic procedures; cardiac surgery and anesthesia; treatment of heart failure, shock, and hypertension; special problems of etiology and therapy; and other subjects of current interest are scheduled for consideration. The course will provide twenty-five hours of formal credit by the American Academy of General Practice.

The registration fee of $25 may be sent to the University of Texas Postgraduate School of Medicine, 1400 Outer Belt Drive, Houston 25, from which additional information also may be obtained.

Wichita Falls Tumor Conference

The third annual State Tumor Conference sponsored by the Wichita County Medical Society Tumor Clinic, the American Cancer Society, and the State Department of Health will be held March 13 in the main auditorium of Midwestern University in Wichita Falls. This clinic, which is approved for credit in formal postgraduate training, is open to physicians without payment of a registration fee.

The program follows:

Address of Welcome—Dr. James T. Lee, Wichita Falls, President of Wichita County Medical Society.
Diagnosis and Treatment of Cancer of Thyroid—Dr. Shields Warren, Boston, New England Deaconess Hospital.
Early Diagnosis and Treatment of Cancer of Stomach—Dr. Alton Ochsner, New Orleans, Tulane University School of Medicine.
Cancer in General Practice—Dr. Jesshill Love, Louisville, St. Joseph Infirmary.
Radioisotopes in Treatment of Tumors—Dr. Warren.
Early Diagnosis of Cancer of Lung—Dr. Ochsner.
Treatment of Malignant Disease—Dr. Love.

Dr. Bailey R. Collins, director of the clinic, will preside over the morning session; Dr. David H. Allen over the afternoon session. Luncheon at the Midwestern University Cafeteria and morning and afternoon recesses are on the schedule, and the day will be ended with a question and answer period.

INTERNATIONAL MEDICAL MEETINGS

Announcements have been made of several international meetings of medical interest:

Congress of Pan American Association of Ophthalmology, Brazilian Congress of Ophthalmology, and *International Congress of Oto-Neuro-Ophthalmology,* June 17-21, Sao Paulo, Brazil. Interested physicians are invited to participate in these three events for a registration fee of $10, which also will cover a number of social events planned in connection with Sao Paulo's four hundredth anniversary. Dr. Daniel B. Kirby, 780 Park Avenue, New York, United States representative on the council of the Pan American Association, can give further details.

International Cancer Congress, July 23-29, Sao Paulo, Brazil. Interested physicians may attend this sixth congress sponsored by the International Union Against Cancer, at which fundamental research, clinical studies, and control of cancer will be considered. Travel by ship and plane and special South American tours are being arranged for visitors from the United States, and financial assistance to cover part of the travel costs is available to a limited number. Further details may be obtained from the Chairman, National Committee on the International Union Against Cancer, National Research Council, 2101 Constitution Avenue, N. W., Washington 25, D. C.

International Congress of Internal Medicine, September 15-18, Stockholm, Sweden. The International Society of Internal Medicine, representing forty nations, will sponsor a program including symposiums on hypertension and collagen diseases and a variety of other papers. Visits to hospitals and scientific institutions are on the agenda. Professor Nanna Svartz, Caroline Medical Institute, Stockholm, is chairman of the planning committee and president of the society.

International Congress on Diseases of the Chest, October 4-8, Barcelona, Spain. All aspects of chest diseases, including cardiorespiratory function and inhalation therapy, will be covered in this world congress. The program will include papers, motion pictures, and scientific and technical exhibits. Murray Kornfeld, executive director of the American College of Chest Physicians, the sponsoring agency, has additional information and may be addressed at 112 East Chestnut, Chicago 11.

MISSISSIPPI VALLEY ESSAY CONTEST

A $100 cash prize, a gold medal, and a certificate are being offered for the best unpublished essay on any subject of general medical interest (including medical economics and education) and of practical value to the general practitioner by the Mississippi Valley Medical Society. Resident citizens of the United States who are members of the American Medical Association may compete. Manuscripts up to 5,000 words must be submitted by May 1. Further information is available from Dr. Harold Swanberg, Secretary, Mississippi Valley Medical Society, 209-224 W.C.U. Building, Quincy, Ill.

UNIVERSITY OF TEXAS MEDICAL BRANCH

December visitors at the University of Texas Medical Branch, Galveston, included David E. Green, Ph. D., director of the Enzyme Institute, University of Wisconsin, Madison; Burton L. Baker, Ph. D., University of Michigan, Ann Arbor; and William C. Rose, Ph. D., professor of biochemistry and nutrition, University of Illinois, Urbana.

Dr. Harriet Felton, professor of pediatrics at the Medical Branch, presented an exhibit on her work to develop a new vaccine for whooping cough at the recent meeting of the American Academy of Pediatrics at Miami.

PRIVATE CLINICS AND HOSPITALS OF TEXAS

Dr. W. R. Swanson, Taylor, assumed duties as president of the Private Clinics and Hospitals Association of Texas at the group's annual meeting December 4-6 in Austin. Other officers are Drs. Neil Buie, Marlin, president-elect; R. D. Holt, Meridian, vice-president; John Dupree, Levelland, secretary-treasurer; and William Blair, Wharton, editor-in-chief.

The program included round-table discussions of problems of privately owned clinics and hospitals in such areas as nursing care, insurance, food, and laboratory service. Leaders of the discussions were Drs. Harvey Renger, Hallettsville; J. P. Anderson, Brady; Van D. Goodall, Clifton; Dupree; Buie; Gilbert Casey, Jacksonville; and Kline S. Dewees, Wills Point. Dr. Joe Nichols, Atlanta; William R. Hunt, Waco; and Bill Burton, El Paso, spoke at the luncheons, and Dr. S. D. Coleman, Navasota, gave the president's address.

SOUTHWESTERN MEDICAL SCHOOL

Dr. William F. Mengert, head of the Department of Obstetrics and Gynecology at Southwestern Medical School of the University of Texas, Dallas, became president of the American Academy of Obstetrics and Gynecology at its meeting in Cincinnati on December 14.

Dr. Hans Selye, director of the Institute of Experimental Medicine and Surgery at the University of Montreal, delivered the Phi Delta Epsilon lecture at Southwestern Medical School of the University of Texas, Dallas, on October 24, using as his subject "The General Adaptation Syndrome."

S. Edward Sulkin, Ph. D., professor of bacteriology at Southwestern Medical School of the University of Texas, Dallas, joined researchers from five countries at the International Symposium on Dynamics of Virus Infections October 21-23 in Detroit.

SOUTHWESTERN MEDICAL ASSOCIATION

Dr. Willard W. Schuessler, El Paso, was installed as president of the Southwestern Medical Association at its thirty-fifth annual meeting October 29-31 in Tucson, Ariz. Other officers elected by the 260 physicians present from Arizona, New Mexico, West Texas, and Northern Mexico follow: Dr. Joseph Bank, Phoenix, president-elect; Dr. N. K. Thomas, Tucson, first vice-president; Dr. John H. Dettweiler, Albuquerque, second vice-president; Dr. Leslie Daviet, Las Cruces, N. M., third vice-president; and Dr. Celso C. Stapp, El Paso, secretary-treasurer.

On the scientific program were Drs. Merl J. Carson, Glendale, Calif.; Donovan C. Browne, New Orleans; Edmund B. Spaeth, Philadelphia; William Paul Thompson, Los Angeles; and Henry K. Ransom, Ann Arbor, Mich.

Baylor Hospital Celebrates Half Century

Baylor Hospital in Dallas on October 16 celebrated fifty years of being chartered by the State of Texas as a hospital. Observance of the occasion included a five-day series of tours for visitors, a banquet with United States Senator Price Daniel as chief speaker, coffees honoring Baylor Nurses' Alumnae and the Baylor Woman's Auxiliary, and dinners for employees and for the medical staff.

Harris County General Practitioners Elect

The Harris County Academy of General Practice has announced the election of officers for 1954 as follows: Dr. Donald M. Gready, president; Dr. William F. Cole, president-elect; Dr. Milton Littell, vice-president; Dr. Ben Walpole, secretary; Dr. Homer A. Taylor, treasurer; Dr. A. C. Madsen, director.

LIBRARY SECTION

GLAUCOMA

To avoid the easy error of assuming we are racing along abreast of an always fast and sure current of medical progress, a deliberate backward look can be sobering. Consider that there exists a twenty-three century old description of glaucoma, that it was some four centuries after this description before any difference between glaucoma and cataract was hinted by Rufus, and that another six hundred years elapsed before a written account by Paulus Aegineta of actual symptomatic differences was made. Aegineta, incidentally, remarked, "Glaucoma is in every case incurable," an opinion repeated as late as 1858 by Desmarres. Consider that the entire Greek-Roman period and the Dark Ages provided little more than recognition of the disease, failing adequately to separate it from cataract, and that this state of affairs continued approximately the same until the appearance of a book by Brisseau in 1709. Consider the remarkable fact that although a condition of firmness or hardness of the glaucomatous eye was recognized in 1622 by Barrister, more than a hundred years passed before general appreciation of the requirement of increased intraocular pressure followed the description of the association by Platner in 1745 and again particularly by Bowman in 1858. Indeed, remember that it was some twenty-two centuries after the Hippocratic description before one appeared by Weller in 1826 which was substantially correct and useful even by today's standards.

The stream of medicine quickened in the nineteenth century, and ophthalmology was no exception. Largely because of von Helmholz's ophthalmoscope, there developed almost automatically a voluminous literature about glaucoma and other subjects. Small, slowing eddies appeared, as evidenced by the misconception about the optic disk in glaucoma; Jaeger and even von Graefe believed it bulged forward rather than being cupped. That giant of ophthalmology, von Graefe, whose incalculably valuable operation (iridectomy) was announced when he was but 22 years of age, required some time before he was completely convinced that hardness of the eyeball was the cause of blindness, since he required proof of concavity of the nerve head. Iridectomy initially seemed phenomenally successful and was almost the sole surgical attack for the subsequent fifty years, although its mode of action was by no means understood for most of that period, even by the originator. Von Graefe felt that every practitioner should be able to diagnose glaucoma, still a desideratum as evidenced by the motion picture film, "Glaucoma: What the General Practitioner Should Know," now available at the Texas Medical Association Memorial Library.

Consider that miotics were introduced as late as 1877 and that the palpating finger estimation of intraocular pressure or tension was only replaced following the announcement of Schiøtz's tonometer in the period 1905-1910. And recall that it is really of only late years that we at last seem able to be categorical about some of the types of glaucoma. In any case, it is the recollection of what has gone before—and the time it took—that will cause a natural hesitancy when it comes to predicting which of more recent observations shall prove lastingly helpful or astonishingly neglected. Yet, there would seem no way any physician can reasonably fail to heed modern words such as these of Kirby:

"Patients, unguided and unhelped, or treated by persons not trained in the detection, recognition and modern treatment of glaucoma, are inevitably destined for blindness. With early recognition, proper treatment and supervision, the condition is serious but the prospects for recovery are usually excellent and useful vision can be maintained. A knowledge of the early symptoms of glaucoma is essential so that timely, remedial measures may be taken while they are still of great value."

Now Texas physicians easily can obtain the previously mentioned film about glaucoma produced by the National Society for the Prevention of Blindness, Inc. From it, we may all be assisted in "early recognition, proper treatment and supervision," thus to keep abreast of the current of medicine, whatever its present speed of movement.

—SAM N. KEY, JR., M. D., Austin.

BOOKS RECEIVED IN DECEMBER

Adler, Francis Heed: *Physiology of the Eye, Clinical Applications,* ed. 2, St. Louis, C. V. Mosby, 1953.

Association for Research in Nervous and Mental Diseases: *Patterns of Organization in the Central Nervous System,* Baltimore, Williams and Wilkins, 1952.

Association for Research in Nervous and Mental Disease: *Psychiatric Treatment,* Baltimore, Williams and Wilkins, 1953.

Bakwin, Harry, and Bakwin, Ruth Morris: *Clinical Management of Behavior Disorders in Children,* Philadelphia, W. B. Saunders, 1953.

Barsky, Arthur Joseph: *Principles and Practice of Plastic Surgery,* Baltimore, Williams and Wilkins, 1950.

Bisch, Louis E.: *Cure Your Nerves Yourself,* New York, Wilfred Funk, 1953.

Brailsforth, James F.: *Radiology of Bones and Joints,* ed. 5, Baltimore, Williams and Wilkins, 1953.

Brams, William A.: *Managing Your Coronary,* Philadelphia, J. B. Lippincott, 1953.

Clinical ACTH Conference Proceedings, Second, Philadelphia, Blakiston, 1950.

Conference on Drug Addiction Among Adolescents, New York, Blakiston, 1953.

Dickinson, Robert Latou: *Human Sex Anatomy,* ed. 2, Baltimore, Williams and Wilkins, 1949.

Engle, Earl T.: *Menstruation and Its Disorders, Proceedings of the Conference,* Springfield, Ill., Charles C Thomas, 1950.

Glaister, John: *Medical Jurisprudence and Toxicology,* Edinburgh, E. & S. Livingstone, 1953.

Gordon, I.; Turner, R.; and Price, T. W.: *Medical Jurisprudence,* Edinburgh, E. & S. Livingstone, 1953.

Haymaker, Webb, and Woodhall, Barnes: *Peripheral Nerve Injuries,* ed. 2, Philadelphia, W. B. Saunders, 1953.

Klendshoj, Niels C.: *Fundamentals of Biochemistry in Clinical Medicine, Springfield, Ill.,* Charles C Thomas, 1953.

Koch, Frederick C., and Hanke, Martin E.: *Practical Methods in Biochemistry,* ed. 6, Baltimore, Williams and Wilkins, 1953.

Mackie, T. J., and McCartney, J. E.: *Handbook of Practical Bacteriology,* ed. 9, Edinburgh, E. & S. Livingstone, 1953.

Means, J. H.: *The Thyroid and Its Diseases,* ed. 2, Philadelphia, J. B. Lippincott, 1948.

Moore, Daniel C.: *Regional Block,* Springfield, Ill., Charles C Thomas, 1953.

Noble, Frank W.: *Electrical Methods of Blood-Pressure Recording,* Springfield, Ill., Charles C Thomas, 1953.

Noyes, Arthur P.: *Modern Clinical Psychiatry,* ed. 4, Philadelphia, W. B. Saunders, 1953.

Parsons, Langdon, and Ulfelder, Howard: *An Atlas of Pelvic Operations,* Philadelphia, W. B. Saunders, 1953.

Puestow, Charles B.: *Surgery of the Biliary Tract, Pancreas and Spleen,* Chicago, Year Book Publishers, 1953.

Russ, Joseph D.: *Resuscitation of the New Born,* Springfield, Ill., Charles C Thomas, 1953.

Scott, Wendell G.: *Planning Guide for Radiologic Installations,* Chicago, Year Book Publishers, 1953.

Seed, Lindon, and Fields, Theodore: *Treatment of Toxic Goiter with Radioactive Iodine,* Springfield, Ill., Charles C Thomas, 1953.

Simons, R. D. G. Ph.: *Handbook of Tropical Dermatology,* Amsterdam, Elsevier, 1952.

Smul, Josef S.: *Respiratory Diseases and Allergy,* New York, Medical Library Company, 1953.

Spurling, R. Glen: *Lesions of the Lumbar Intervertebral Disc,* Springfield, Ill., Charles C Thomas, 1953.

Weil, Alfred J., and Saphra, Ivan: *Salmonellae and Shigellae,* Springfield, Ill., Charles C Thomas, 1953.

Weisberg, Harry F.: *Water, Electrolyte and Acid-Base Balance,* Baltimore, Williams and Wilkins, 1953.

Wolstenholme, G. E. W.: *Ciba Foundation Symposium, Visceral Circulation,* Boston, Little, Brown and Co., 1953.

Zimmerman, Leo M., and Anson, Barry J.: *Anatomy and Surgery of Hernia,* Baltimore, Williams and Wilkins, 1953.

CONTRIBUTIONS TO LIBRARY

Grateful acknowledgment is made by the Texas Medical Association Memorial Library of the following recent gifts:

Dr. T. J. Archer, Austin, 95 journals.

Dr. Robert Franken, Austin, 2 journals.

Dr. P. I. Nixon, San Antonio, "Medical Ethics," 1803, by Thomas Percival.

Dr. Morris Polsky, Austin, 130 journals.

Dr. Henry Renfert, Austin, 1 book.

Dr. Earl L. Yeakel, Jr., Austin, 10 journals.

Unusual Gift to Library

Dr. E. E. Lowrey, Gatesville, has given the Library an anatomic model of the skull. The model has a hinged jaw, and the calvarium is cut and may be removed. Made of hard, rigid ivory plastic, the realistic skull has all vascular and neural openings present, and the bone structure and sutures are reproduced.

MOTION PICTURES FOR LOAN

Drug Addiction

16 mm., sound, 21 minutes. (Purchased by Texas Medical Association Memorial Library.)

This is a dramatic and straightforward story of the hazards of narcotic drugs. It depicts with stark realism all phases of this problem, particularly as it affects teenagers. The film explains the derivation of heroin, marijuana, and cocaine and employs animated drawings to describe their physiologic effects. The connection between drug addiction and crime is noted, and the highlights of a typical case are dramatized.

Surgical Treatment for Carcinoma of Lower End of Esophagus

16 mm., silent, color, 42 minutes. (Courtesy of Dr. Philip Thorek, Chicago.)

This film presents a case in which carcinoma of the cardiac end of the stomach extends into the esophagus. A transthoracic partial esophagogastrectomy is performed with an end-to-side esophagogastrostomy. Roentgen-ray studies and the patient's postoperative course are included.

Hemicolectomy for Carcinoma of Right Side of Colon

16 mm., silent, color, 22 minutes. (Courtesy of Dr. Philip Thorek, Chicago.)

Surgical treatment for adenocarcinoma of the ascending colon is presented in the film. Following mobilization of the ascending colon in the proper avascular cleavage plane, the last 6 inches of ileum, cecum, ascending colon, hepatic flexure, and proximal half of the transverse colon are removed. An aseptic end-to-end ileo transverse colostomy is performed. Preoperative and postoperative roentgenograms and the patient one year postoperatively are shown.

Treatment of Varicose Veins

16 mm., silent, color, 30 minutes. (Courtesy of G. D. Searle and Company.)

This film includes a discussion of the venous anatomy of the leg, demonstrates by animation the various types of varicosities, and shows the treatment of each type. A test to determine the adequacy of collateral circulation is demonstrated. This is an excellent film for general practitioners and surgeons.

What Is Cancer?

16 mm., sound, color, 30 minutes. (Courtesy of Texas Cancer Coordinating Council, Austin.)

Designed especially for nurses and nursing trainees, this film is helpful in explaining the problem of cancer from a nurse's standpoint. This film is excellent for nurses and hospital groups. Physicians concerned with nursing education programs will find this a valuable adjunct to their lectures.

BOOK NOTICES

[1]Introduction to Physiological Optics

ARMIN VON TSCHERMAK-SEYSENEGG, *University of Munich, Regensburg Extension. Translation sponsored by the American Committee on Optics and Visual Physiology of the Section on Ophthalmology, American Medical Association; American Ophthalmological Society; and American Academy of Ophthalmology and Otolaryngology. Translated by* PAUL BOEDER, PH. D., *Instructor in Ophthalmology, Harvard Medical School, 298 pages. $10.50. Springfield, Ill., Charles C Thomas, 1952.*

The student who looks for information on physiology of vision finds relatively scanty information in the English ophthalmic and physiologic literature. Hence this monograph is an effort to arouse primarily an interest of larger ophthalmologic groups in physiologic optics and to invite them to practical evaluation. It is a masterly survey of the physiology of vision, especially in the physiology of binocular vision and ocular motility.

The book is divided into six chapters, the first dealing with the optic image, its significance and formulation. The second deals with an introduction to the light sense, photometric and physiologic factors. This stresses that according to the method of exact subjectivism, the treatment of the light-color sense must not begin with the physical discussion of the light stimulus but with the analysis of the physiologic effect. This basic conception opposes the older objectivistic conception which asserted direct perception through the sensory tools or at least a simple causal connection between stimulus and effect.

Following this is an introduction to color sense, giving a

[1]*J. W. Pickens, M. D., Cleburne.*

general description of the character of color sensations, relationship of wave length, intensity, and composition of light stimuli, and the qualities of color sensation.

The latter part of this monograph discusses binocular space sense and the physiology of ocular movements, giving special attention to retinal correspondence and to the laws governing the movements of the eye.

The author of the book concludes that "In spite of the unavoidable incompleteness of the presentation and in spite of the perhaps somewhat strongly subjective coloring, . . . the harmony of the resulting picture should not be overlooked."

[2]Office Psychiatry

LOUIS G. MOENCH, M. D., *Assistant Clinical Professor of Medicine and of Psychiatry, University of Utah School of Medicine. 310 pages. $6. Chicago, Year Book Publishers, 1952.*

This is an exceptionally fine book in that it accomplishes well the author's desire of acquainting the general practitioner, the nonpsychiatric specialist, and the medical student with the more valued contributions of psychiatry to the practice of medicine.

The book is comprehensive in scope but leaves out all of the argumentative and controversial afflictions of psychiatry. At times oversimplification might seem to defeat the author's purpose, but on the whole probably it will stimulate the readers to investigation of the references in his bibliography.

I was impressed particularly with the introductory chapters and the chapter entitled "The Interview."

Throughout the text there are numerous caricatural illustrations that cleverly emphasize important interpersonal situations and possibly some pet peeves of the author.

A combination of excellent writing technique with adequate understanding of the prospective readers definitely increases the value of this contribution.

[3]Contact Dermatitis

GEORGE L. WALDBOTT, M. D., F.A.C.P., *Senior Physician, Harper Hospital; Chief of Division of Allergy, Grace Hospital; Chief of Allergy Clinic, Children's Hospital of Michigan, Detroit. Edited by* ARTHUR C. CURTIS, M. D., *Chairman, Department of Dermatology and Syphilology, University of Michigan, Ann Arbor. A Monograph in the Bannerston Division of American Lectures in Dermatology. 218 pages. $8.75. Springfield, Ill., Charles C Thomas, Publisher, 1953.*

The interrelated fields of allergy and dermatology are correlated in this work on a dermatologic subject by an allergist. It is a striking departure from the usual "cut and dried" style of the ordinary textbook, and it makes good reading.

The book covers incidence, mechanism, pathology, and diagnosis in an abbreviated form, and these chapters present no unusual features. One still must be familiar with clinical dermatology to diagnose contact dermatitis. The real value of the book appears in the chapters devoted to determination of the causes and "the pattern." The author has devised a method of marking the exposed surfaces of skin in such a way as to denote the contact areas and to group the patterns according to the causes, as, for instance, the "reaching into" pattern, the "finger grasp" pattern, and the "large ball" or "small ball" patterns. By referring to the pattern of a dermatitis, the detective job of the clinician is greatly aided, and clues are ascertained by which deductions may be made, the appropriate tests performed, and the guilty antigen may be indicated.

In a section on "Special Situations," plants, pollen, cosmetics, soap, wearing apparel, industrial irritants, drugs, and the like are discussed as sources of dermatitis. The author rightly evaluates the psychosomatic aspect of skin diseases, notably disposing of "nervousness" as the cause for every otherwise unexplainable condition.

Therapy of allergic dermatoses is well covered, and the point of avoidance of further aggravation by ill chosen medicaments is well taken.

The appendices, tables, and bibliography are good. The author has indulged in worthy consultations, and his editor, a dermatologist of note, was well chosen. There is a glossary inserted for the benefit of "non-medical readers." Undoubtedly, this book will be read by many such readers, but whether or not the glossary will enable them to place the proper interpretation upon the subject matter is, to this observer, highly problematical.

[4]Gifford's Textbook of Ophthalmology

FRANCIS HEED ADLER, M. D., *Professor of Ophthalmology, University of Pennsylvania Medical School; Consulting Surgeon, Wills Eye Hospital, Philadelphia. Fifth edition. $7.50. Philadelphia, W. B. Saunders Company, 1953.*

This work is designed primarily for the instruction of undergraduates in medicine and for physicians not specializing in the practice of ophthalmology.

Each of the twenty-two chapters contains a thorough discussion of the subject to which it pertains. The anatomy and physiology of the various parts and structures are given. Aside from an occasional mention, surgical techniques are entirely eliminated. Treatment of the various ocular diseases and injuries (other than surgical) is presented quite extensively. Hypertensive diseases and diabetes are presented in more than average detail.

The entire contents of this edition are presented in a clear, concise but comprehensive manner, with superfluous details and verbiage eliminated.

This is a textbook that should be of ultimate value to the undergraduate student of medicine, to practitioners, and most particularly to those engaged in general practice. It will be a valuable adjunct to every medical library.

[5]Hypersplenism—A Clinical Evaluation

CYRUS C. STURGIS, M. D., *Chairman of the Department of Internal Medicine and Director of the Thomas Henry Simpson Memorial Institute for Medical Research, University of Michigan. A Monograph in American Lectures in Medicine. 97 pages. $3.25. Springfield, Ill., Charles C Thomas, 1953.*

This monograph is the William Beaumont Lecture delivered to the Wayne County (Michigan) Medical Society in January, 1952. The entire problem of hypersplenism is presented with a review of the literature in a concise and unbiased manner. The subject is defined and a summary of the current ideas concerning the functions and circulation of the spleen is included. The possible mechanisms of the hypersplenic state are discussed, following which the author classifies and discusses the types of hypersplenism specifically as regards etiology, pathology, signs and symptoms, laboratory findings, diagnosis, and treatment. Occasional case histories with the charted laboratory findings serve to enhance the value of the text. The bibliography and index are adequate. All in all, this monograph is a satisfactory compendium of the general problem as it is understood today. It can be recommended to medical students, general practitioners, and internists.

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[2]*F. T. Harrington, M. D., Dallas.*
[3]*Evan B. Hume, M. D., Brownsville.*
[4]*Frederic H. Thorne, M. D., Kerrville.*
[5]*G. N. Irvine, Jr., M. D., San Angelo.*

Clinical Diagnosis by Laboratory Methods

JAMES CAMPBELL TODD, Ph. B., M. D., *Late Professor of Clinical Pathology, University of Colorado School of Medicine, Denver;* ARTHUR HAWLEY SANFORD, M. D., *Emeritus Professor of Clinical Pathology, The Mayo Foundation, University of Minnesota, Minneapolis; Emeritus Member, Division of Clinical Laboratories, The Mayo Clinic, Rochester; Director of Laboratories, Rochester State Hospital; and* BENJAMIN B. WELLS, M. D., Ph. D., *Professor of Medicine, Department of Medicine, School of Medicine, University of Arkansas, Little Rock. Twelfth edition. 998 pages. $8.50. Philadelphia, W. B. Saunders Company, 1953.*

The new edition of a standard textbook in medicine has been brought up to date, with the section on hematology being rewritten and many new additions to the chapter on serology being made. Each test is explained step by step and many tests are so simplified that they can be performed in the office without any special equipment. Every possible result of each test has been considered, and the significance of each in the clinical picture is given.

The index outline of laboratory findings arranged in alphabetical order adds to the value of this well known reference tool.

[6]Physiological and Therapeutic Effects of Corticotropin (ACTH) and Cortisone

DWIGHT J. INGLE, Ph. D., *Senior Research Scientist (Physiologist), The Upjohn Company, Kalamazoo, Mich.; and* BURTON L. BAKER, Ph. D., *Professor of Anatomy, University of Michigan Medical School, Ann Arbor. A Monograph in American Lectures in Metabolism. 172 pages. $5.50. Springfield, Ill., Charles C Thomas, 1953.*

This monograph is an effort on the part of two research scientists to compile all the known information about the adrenal cortical hormones in a readable form for the average physician. The first seventy-five pages deal with the nature of the various hormones and with their effects on normal and abnormal tissue. They show step by step how work regarding these hormones is carried out on experimental animals, on persons with Addison's disease, and in various other disease processes. Diseases in which the hormones have been used are discussed. Beneficial results, where obtained, are described and weighed against the undesirable side effects in an effort to determine their actual value.

This book is not written with a viewpoint of being used in day to day practice but rather to give the reader a good general idea about the whole field in a few pages. An extensive bibliography covering thirty-one pages is found at the end of the book. I found it a very interesting book to read, but I doubt its value in the average physician's library.

[7]Hormonal and Neurogenic Cardiovascular Disorders

WILHELM RAAB, M. D., F.A.C.P., F.A.C.C., F.C.C.P., *Professor of Experimental Medicine and Head of Cardiovascular Research Unit, University of Vermont, Burlington. 722 pages. $15. Baltimore, Williams and Wilkins Company, 1953.*

The author's purpose is "to convince cardiologists and other physicians that neuroendocrine and endocrine influences play a fundamental and decisive part in the origin and therapeutic amenability of many cardiovascular disorders." In this he has admirably succeeded.

The book is divided into three main sections: experimental cardiovascular effects of hormones and neurohormones, cardiovascular features in endocrine and neuroendocrine syndromes, and endocrine and neuroendocrine factors in cardiovascular syndromes.

There are helpful summaries of individual chapters. Each section has a synopsis. The book concludes with a general summary and more than 3,000 references to the literature.

The reviewer noted only one error; in Read's formula on page 129 "the pulse rate" should be corrected to "the pulse pressure."

This book is heartily recommended to those interested in the fields of endocrinology and cardiovascular diseases.

[8]Amebiasis: Pathology, Diagnosis and Chemotherapy

HAMILTON H. ANDERSON, M. D., *Professor and Chairman, Pharmacology and Experimental Therapeutics;* WARREN L. BOSTICK, M. D., *Associate Professor of Pathology, Department of Pathology; and* HERBERT G. JOHNSTONE, Ph. D., *Professor of Parasitology, Parasitology and Mycology Section of the Department of Medicine, School of Medicine, University of California Medical Center, San Francisco. 431 pages. $11.50. Springfield, Ill., Charles C Thomas, 1953.*

This book is one of the best that has been brought out in regard to amebiasis. It covers the subject thoroughly, is well planned, and goes into adequate detail. Controversial points are well handled and discussed, and these are presented in an unbiased manner.

The authors make every attempt to do away with the commonly accepted symptom of dysentery as one of the most looked-for symptoms in amebiasis. It has been shown repeatedly that dysentery itself is not infrequently absent in amebiasis, and this fact alone is well worth bringing before the profession as a whole.

The book is divided into convenient chapters, each of which goes thoroughly into its particular aspect of amebiasis, and at the end of each chapter is an extensive bibliography. For the general practitioner, the chapters dealing with symptomatology and clinical manifestations and with laboratory diagnosis of amebiasis are especially well presented.

[9]Clinical Cardiology

Edited by FRANKLIN C. MASSEY, A. B., M. D., *Assistant Professor of Medicine, Hahnemann Medical College, Philadelphia. 1,060 pages. $13.50. Baltimore, Williams and Wilkins Company, 1953.*

This book contains several excellent chapters, but as a whole it does not compare favorably with other available textbooks of cardiology. Written by thirty-three collaborators, it lacks that warm, almost personal relationship that should be obtained between author and reader. In addition, while the editor admits in his preface that multiple authorship often involves poor continuity and perspective, he has not always offset these defects by choosing contributors of invariably high standards of scholarship and experience.

The chapter on surgery of the heart is especially commendable. Also authoritative and readable are the sections on fundamentals (as opposed to clinical aspects) of coronary circulation, endocarditis, and roentgenology. Electrocardiography is reliably presented but too compressed and the illustrations too few. Schemm's discussion of congestive failure is stimulating, though his understandable enthusiasm for his own ideas has led him to slight other useful therapeutic measures. Illustrations in the book range from strikingly beautiful to inartistic, and many of the bibliographies are incomplete.

Misleading errors of fact are numerous. That intensity alone will distinguish functional from organic murmurs is

[6]*W. S. Rhode, M. D., Colorado City.*
[7]*H. L. Wolf, M. D., Amarillo.*
[8]*O. B. Gober, M. D., Temple.*
[9]*Royall M. Calder, M. D., San Antonio.*

debatable and that organic cardiac lesions never alter the first pulmonic sound is patently erroneous. Few clinicians would agree that it is "nonsense" to administer quinidine for the prevention of possibly fatal arrhythmias complicating myocardial infarction or that it is impossible to master the art of percussing the heart. Likewise, believing that uncomplicated neurocirculatory asthenia is often characterized by fever might well lull an immature clinician into overlooking a remedial infection.

Muscle metabolism is ignored and dosages and pharmacology of drugs (even digitalis) often are treated in a sketchy fashion. Ouabain, digitalis glucosides, and many other drugs of established usefulness are not mentioned.

Striving for elegance of style has resulted frequently in declamations rather than simple, informative exposition and in the misuse of big words when little ones would have served. Occasional neologisms, unacceptable abbreviations, mistakes in simple grammar, and innumerable misspelled words bespeak indifferent editing.

This reviewer has read every page of this text, is not a competing author, and is disappointed that the unquestionably good features of this book do not outweigh the bad.

[10]Hypnosis in Modern Medicine

Edited by JEROME M. SCHNECK, M. D., *Clinical Associate in Psychiatry, College of Medicine, State University of New York; Founder and President, Society for Clinical and Experimental Hypnosis. 323 pages. $7.50. Springfield, Ill., Charles C Thomas, 1953.*

This book is composed of eleven separate chapters, each written by a different investigator and each having an extensive bibliography appended to it. Through this type of organization the author has attempted to bring under one cover the experience and knowledge of certain selected authorities who are proficient in their respective areas of interest within the broader field of hypnosis in its relationship to medicine. The book offers interesting information on the history of hypnosis and its use in various of the specialties of medicine, surgery, and dentistry.

There is an especially interesting chapter on the physiologic aspects of hypnosis.

This publication can be read with ease and offers an opportunity for every physician to become more informed on a subject which is both controversial and somewhat neglected in the medical preparation of the average physician.

[11]Physical Examination of the Surgical Patient

J. ENGLEBERT DUNPHY, M. D., F.A.C.S., *Associate Clinical Professor of Surgery, Harvard Medical School; Surgeon, Peter Bent Brigham Hospital, and* THOMAS W. BOTSFORD, M. D., F.A.C.S., *Clinical Associate in Surgery, Harvard Medical School; Senior Associate in Surgery, Peter Bent Brigham Hospital. 326 pages. $7.50. Philadelphia, W. B. Saunders Company, 1953.*

This book will prove to be a needed adjunct in the teaching of physical diagnosis since for some time emphasis has been placed upon the examination of the chest. Most surgical conditions are covered in the text, and practicing physicians will find it helpful in differentiating surgical lesions. Descriptions of the techniques and procedures to be mastered in surgical diagnosis are detailed and well illustrated. In every section the approach to the examination is presented in such a way as to make the doctor-patient relationship more pleasant and the examination easier.

Each system of the body is considered separately, and there is a special chapter on the examination of the abdomen in infants and children. The last six chapters will be of interest to all physicians in general practice since methods are presented for rapid diagnosis following trauma.

Doctor in the House

RICHARD GORDON. *186 pages. $2.75. New York, Harcourt, Brace and Company, 1953.*

"Doctor in the House" is a fantastic story of an English medical school. It is beyond comprehension for anyone to read the story and see such frivolous young men in medical schools today, regardless of the country.

This book is well written and easy to read, yet the impression one receives of the English medical student is certainly not complimentary, and a person can be happy the student will not be his doctor some day.

[10]*Anthony P. Rousos, M. D., Austin.*

[11]*Robert M. Tenery, M. D., Waxahachie.*

ORGANIZATION SECTION

AMERICAN MEDICAL ASSOCIATION

Clinical Session Attended by 7,716

There were 7,716 persons including 2,730 physicians registered for the seventh annual clinical session of the American Medical Association in St. Louis, December 1-4. In addition to a full scientific program, the meeting included sessions of the House of Delegates in which policy action was taken in a number of areas.

The House adopted a modification of the Principles of Medical Ethics which clarifies the relationship of physicians to all form of public information media and referred to the Board of Trustees a resolution calling for the appointment of a special committee to study the publicity problems which result from unethical practices by a small minority of physicians.

Opposition to compulsory coverage of physicians under federal old age and survivors insurance was voiced again, whereas passage of the Jenkins-Keogh bills to permit a voluntary pension program for the self-employed, of the Bricker amendment to restrict treaty-making powers, and of measures to reduce or remove the limitation on the deduction of medical and dental expenses for income tax purposes was approved. The House voiced strong opposition to extension of the doctor draft beyond the present expiration date of June 30, 1955. A resolution endorsing the principle of federally subsidized scholarships for prospective military personnel was referred to the Trustees.

The Council on Medical Service was requested to proceed at once with special study of problems relating to catastrophic coverage and coverage for retired persons in voluntary health insurance plans. The House again affirmed itself as considering pathology, radiology, anesthesiology, and psychiatry as medical services and condemned insurance contracts which classify any such medical service as hospital service. The Joint Commission on Accreditation of Hospitals was requested to publicize its regulations and interpretations on accreditation.

The House referred to the Judicial Council a resolution from Iowa calling for approval of a joint billing procedure when services are rendered by two or more physicians.

Note was made that the American Medical Association has given a fourth grant of $500,000 to the American Medical Education Foundation. The foundation reported a total income for 1953 of $1,174,000 with the number of contributors more than double that for 1952.

One of the speakers before the House of Delegates was Dr. Chester Keefer of Boston, special assistant to the Secretary of Health, Education, and Welfare in Washington.

Dr. Joseph I. Greenwell, New Haven, Ky., was named General Practitioner of the Year.

NEW AMA TELEVISION FILMS

The American Medical Association's Television Film Library now has available four new television shows to be used exclusively by local medical societies. The films will not be aired on any national television network but have been designed to be presented on public service time locally.

The films are "Operation Herbert," which deals with medical care costs; "A Citizen Participates," which shows a citizen's campaign to get a doctor for the town; "What to Do," which is a series of six five-minute shows demonstrating the use of a thermometer, the application of artificial respiration, the stocking of a medicine chest, and emergency treatment of colds, abdominal pains, and headaches; and "Your Doctor," which describes medical training facilities and focuses on the career of a general practitioner in a rural community.

Prints of these films may be obtained by county societies by writing to the AMA's Television Film Library, 535 North Dearborn Street, Chicago 10.

Education and Licensure Congress Set

The annual Congress on Medical Education and Licensure conducted by the Council on Medical Education and Hospitals of the American Medical Association and the Federation of State Medical Boards of the United States will be held February 8 and 9 in Chicago. An open meeting of the Advisory Board for Medical Specialties will be held the preceding day.

Blasingame Resigns Legislative Job

Dr. F. J. L. Blasingame, Wharton, who has been serving as chairman of the Legislative Committee of the American Medical Association, has resigned from that position because of pressure of other commitments. Dr. Blasingame is a member of the AMA Board of Trustees.

Industrial Health Conference at Louisville

The fourteenth annual Congress on Industrial Health sponsored by the Council on Industrial Health of the American Medical Association will convene February 23-25 at Louisville. The role of industry in the maintenance of the health of the nation, health programs for executives, screening examinations, employment welfare of the handicapped, problems of the small plant operator, preparation for emergencies by plant and community, and the relationship of American medicine with labor and management will be considered.

Rural Health Conference in Dallas

The ninth National Conference on Rural Health sponsored by the Council on Rural Health of the American Medical Association will be held at the Baker Hotel, Dallas, March 4-6. Additional details of this conference will appear in the February JOURNAL.

TEXAS MEDICAL ASSOCIATION

MORE TELEPHONE BROADCASTS

Two telephone broadcasts under the sponsorship of the Council on Medical Education and Hospitals of the Texas Medical Association and available to county medical societies or other interested professional groups have been announced for February 16 and March 9. Both broadcasts will originate at the headquarters building of the Texas Medical Association in Austin.

The February program will be presented by members of the faculty of the University of Texas Medical Branch, Galveston, with Dr. George R. Herrmann as moderator. Dr. Herrmann also will present a discussion on "Recent Advances in Cardiovascular Diseases." Other topics and speakers include "Recent Advances in Endocrinology and Metabolism" by Dr. Raymond L. Gregory; "Recent Advances in Hematology" by Dr. William C. Levin; and "Recent Advances in Gastroenterology" by Dr. Edward J. Lefeber.

The March program, for which Dr. Henry M. Winans will be moderator, will be offered by Dallasites. Topics and speakers will be "Diet in Cardiovascular Disease" by Dr. Donald Seldin; "Diet in Metabolic Disease" by Dr. Edwin L. Rippy; and "Diet in Gastrointestinal Disease" by Dr. Tate Miller.

Local Committees Named

Members of the local committees for the 1954 San Antonio annual session of the Texas Medical Association, which is to be held May 3-5, have been announced by Dr. L. Bonham Jones, chairman of the Committee on General Arrangements. Serving with Dr. Jones on this steering committee are the following physicians, all of San Antonio: Drs. Charles L. McGehee, Edward D. Dumas, John M. Smith, Jr., and Robert R. Nixon. Other committees are as follows:

Alumni.—William D. Montgomery, chairman; G. G. Passmore, University of Texas School of Medicine; C. C. Shotts, Baylor University College of Medicine; J. Walter Park, III, Southwestern Medical School of the University of Texas; James E. Pridgen, Tulane University of Louisiana School of Medicine; William M. Center, University of Arkansas School of Medicine; Mildred E. Ward, Louisiana State University School of Medicine.

Fraternity.—William J. Johnson, Jr., chairman; Edwin M. Sykes, Jr., Alpha Kappa Kappa; James E. L. Reveley, Nu Sigma Nu; S. Perry Post, Phi Beta Pi; James L. Mims, Jr., Phi Chi; John L. Matthews, Phi Rho Sigma; William J. Fetzer, Theta Kappa Psi; R. Bernard Pomerantz, Phi Delta Epsilon.

Halls and Lanterns.—Robert R. Nixon, chairman; John P. Heaney, Vernon S. Wier.

Hotels.—John M. Smith, Jr., chairman; Jack B. Lee, Ralph A. Munslow.

Information.—S. Perry Post, chairman; Benjamin B. Shaver, Pat I. Nixon, Jr.

Memorial Services.—B. F. Stout, chairman; J. L. Cochran, P. I. Nixon.

Publicity.—A. F. Clark, Jr., chairman; O. Roger Hollan, Jack M. Partain.

Scientific Exhibits.—Arthur C. Ressmann, chairman; Lawrence M. Shefts, Frank M. Posey, Jr.

Social.—Joseph W. Palmer, chairman; John C. Parsons, John J. DeLeon, William W. Sawtelle, Robert H. Vadheim, William J. Block, Jr.

Sports (Golf).—John B. Case, chairman; John C. Parsons, Arthur B. Pyterek.

Technical Exhibits.—Carl E. Bosshardt, chairman; Robert F. Gossett, Daniel D. Altgelt.

Transportation.—Adolfo Urrutia, chairman; Walter Walthall, Phillip L. Day.

Executive Council to Meet

As announced previously, the Executive Council of the Texas Medical Association will meet at the Association's building in Austin on January 24, beginning at 8:30 a. m. The preceding day a conference for presidents and secretaries of county medical societies will be held at the same place beginning at 10 a. m.

COUNTY SOCIETIES

Andrews-Ector-Midland Counties Society

October 15, 1953

Will Private Practice of Medicine Survive?—F. J. L. Blasingame, Wharton.

Dr. Blasingame, President-Elect of the Texas Medical Association, was principal speaker at a meeting of the Andrews-Ector-Midland Counties Medical Society October 15 in Midland, to which the Howard-Martin-Glasscock-Borden-Scurry-Kent-Dickens-Garza-King-Stonewall Counties Medical Society was invited. He spoke on the above subject.

Bell County Society

October 7, 1953

(Reported by H. H. Brindley, Secretary)

Mediastinal Tumors—G. V. Brindley, Jr., Temple.

After the business session of the October 7 meeting of the Bell County Medical Society, Dr. Brindley presented the above discussion with picture slide illustrations. John Henderson was co-author of the paper. The meeting was held in Temple.

A letter from Mrs. A. Ford Wolf, Temple, chairman of the local auxiliary's essay contest committee, was read, asking if the society again wanted to sponsor the Association of American Physicians and Surgeons essay contest. It was decided that Bell County would not participate this year.

The problem of venereal disease control was discussed and a report given by Harry Frey, Belton, of a meeting of various officials of Temple and of Bell County and Army officers from Fort Hood. J. H. Johnson, Temple, presented a revised report and recommendations by the public health committee, and the report and recommendations as presented were adopted.

Applications for membership of Frederick J. Fay and W. J. Bruce were accepted unanimously.

December 2, 1953

(Reported by H. H. Brindley, Secretary)

Mechanisms of Pain in Duodenal Ulcer—N. C. Hightower, Temple. Discussion—A. C. Broders, Jr., Temple.

The Bell County Medical Society met in Temple December 2 to hear the above scientific program and to elect officers. New officers are Hanes H. Brindley, president; Joe A. Fowler, vice-president; Fred M. Hammond, secretary; R. R. Curtis, J. B. Talley, and J. W. Pittman, censors; R. A. Murray and James C. Walker, delegates; and R. N. Bartels and Ralph Greenlee, alternate delegates. Dr. Fowler is from Killeen; Dr. Pittman is from Belton; all other officers are from Temple.

Dr. Curtis stressed the importance of the American Medical Education Foundation and the responsibility of the doctors in connection with the foundation.

The society voted to give its secretary's secretary $100 for her assistance to the society during the year.

Bexar County Society

December 8, 1953

(Reported by Catherine P. Chester, Executive Secretary)

The following officers of the Bexar County Medical Society were installed December 8 at the San Antonio Country Club: Merton M. Minter, president; Robert F. Gossett, president-elect; C. W. Tennison, vice-president; O. R. Hollan, secretary; Daniel D. Altgelt, treasurer; Charles L. McGehee, trustee; and E. W. Coyle, censor. J. Lewis Pipkin is delegate and Ralph A. Munslow, alternate. All officers are from San Antonio. Dr. Coyle, outgoing president, was installing officer.

Speaker at the dinner was Mr. C. R. Spearman, president of the San Antonio Chamber of Commerce, who presented an outsider's viewpoint of "A Doctor's Responsibility to His Community." Chairmen of the society's various committees reported on committee activities for the year.

Cameron-Willacy Counties Society

November 16, 1953

(Reported by David F. Nickell, Executive Secretary)

Clinical Significance of Intraglandular Thyroid Carcinoma—William O. Russell, Houston.

The above scientific program was presented in Brownsville at the November 16 meeting of the Cameron-Willacy Counties Medical Society after a social hour and dinner with the society's woman's auxiliary.

The group voted to write notes of thanks to the *Valley Morning Star* and the *Brownsville Herald* for their coverage of Texas Medical Association's one hundredth anniversary and to add copies of the special editions of both papers to the society's permanent records. Members who assisted in the publication of the editions also were thanked.

W. M. Hibbitts, Texarkana, was a guest at the meeting.

December 14, 1953

(Reported by David F. Nickell, Executive Secretary)

After the annual Christmas party of the Cameron-Willacy Counties Medical Society and its auxiliary in Raymondville December 14, the society held a business meeting and elected officers. J. C. George, Brownsville, became president; David F. Nickell, Harlingen, vice-president; Harry A. Miller, Brownsville, secretary-treasurer; Hunter Scales, San Benito, delegate; and Thurman Kinder, Brownsville, censor.

The society voted to pay each year the basic expenses incurred on the delegate's trip to the Texas Medical Association's annual session and also to contribute $150 yearly to the auxiliary's nurse scholarship fund.

Dudley Smith, Harlingen, president, presided over the business meeting, and Charles Spence, Raymondville, acted as master of ceremonies for the dinner party. Marcel Bedri, Harlingen, played a violin solo, accompanied by Mrs. Herbert Strass, Harlingen. Mrs. Martin Atchison, Harlingen, reported on the auxiliary's nurse recruitment program.

Approximately seventy members and their wives attended.

Dallas County Society

November 10, 1953

(Reported by W. W. Fowler, Secretary)

Hope for Control of Carcinoma of Breast—C. D. Haagensen, New York.

The Dallas County Medical Society met November 10 at Dallas. Dr. Haagensen, who is associate professor of surgery at Columbia University College of Physicians and Surgeons, presented the fourth annual Charles M. Rosser Lecture.

Jack G. Kerr, president, then conducted a business session, during which the nominating committee, with Charles L. Martin as chairman, submitted a report.

Ona Mae Austin, Thomas G. Price, and Jerry Culwell were elected to membership; and the society approved the proposal by which the Texas State College for Women, Denton, would take over the school of nursing conducted by Parkland Hospital. Elliott Mendenhall requested that society members renew their subscriptions to *Today's Health* through the local woman's auxiliary.

Gonzales County Society

December 2, 1953

(Reported by James C. Price, Secretary)

The Gonzales County Medical Society met for dinner and a business session December 2 in Gonzales to hear the Texas Medical Association's telephone postgraduate broadcast on "Obstetric Emergencies" and to elect officers. The 1954 officers are Henry S. Edwards, Nixon, president; Walter A. Sievers, Gonzales, vice-president; and James C. Price, Gonzales, secretary.

Grayson County Society

October 13, 1953

Roentgen-Ray Problems—Arthur Boyd, Sherman.
Tumor Clinic—Philip Montgomery, James Martin, Billie L. Aronoff, James Howard, and E. K. Siebel, Dallas.
Case Presentations—Stanley Clayton, T. A. Moorman, and Maurice Weisberg, Denison.

The three-point program outlined above was given at the monthly dinner meeting of the Grayson County Medical Society October 13 at the Woodlawn Country Club in Denison.

Gregg County Society

October 13, 1953

Peptic Ulcer—A. C. Broders, Jr., R. R. White, and A. W. Sommer, Temple.

The above staff members of Scott and White Clinic presented a panel discussion on peptic ulcer at the October 13 meeting of the Gregg County Medical Society in Longview.

Howard-Martin-Glasscock-Borden-Scurry-Kent-Dickens-Garza-King-Stonewall Counties Society

October 27, 1953

Care of Major Head Injuries in General Practice—J. Victor Ellis, Amarillo.

Members of the staff of the Big Spring State Hospital were hosts to the Howard-Martin-Glasscock-Borden-Scurry-Kent-Dickens-Garza-King-Stonewall Counties Medical Society for a dinner meeting October 27 in Big Spring. The above scientific discussion was given, followed by a question and answer period.

Roy Sloan, Big Spring, presided at the dinner session; he introduced guests including James Bethea and Sam Hoerster, Jr., both associated with the Board for Texas State Hospitals and Special Schools, Austin.

The local woman's auxiliary met with the society for dinner, followed by a separate business session.

Lubbock-Crosby Counties Society

November 2, 1953

(Reported by Frank C. Goodwin, Secretary)

Hip Fractures and Prostheses—John J. Hinchey, San Antonio.
Discussion—Richard Lewis, Lubbock; Harry Tubbs, Post; and Charles Ratcliff, Lubbock.

Wallace I. Hess, president, presided over the November 2 meeting of the Lubbock-Crosby Counties Medical Society in Lubbock.

A letter from J. A. Chatman asking the reaction of the medical society to presenting a five-hour scientific program at a forthcoming state meeting of the Lone Star State Medical Association in Lubbock was read, and the society was in favor of the proposal. The society also accepted Dr. Chatman's invitation to hold its December meeting at the Chatman Clinic-Hospital.

It was announced that the revised constitution and bylaws, which had been approved by Mr. Philip Overton, legal counsel for the Texas Medical Association, will be mimeographed and sent to the society members.

The society voted to adopt the recommendation of the executive board to hold an annual banquet after the first of the year to introduce new officers of the society and of the woman's auxiliary and to foster fellowship among the members.

The society endorsed the proposed project of the local chapter of the Texas Heart Association for checking pulse and blood pressure by registered nurses. Upon request of the president of the District 3 Medical Society, the local society voted to sponsor a speaker for the section on obstetrics and gynecology at the next district meeting.

Nominations were made for officers to be elected at the next meeting, and the above scientific program was presented.

Milam County Society

October 13, 1953

(Reported by Marvin H. Bartlett, Secretary)

The Milam County Medical Society met October 13 in Cameron and elected the following officers for 1954: Thomas S. Barkley, Rockdale, president; John T. Richards, Rockdale, vice-president and delegate; and Marvin H. Bartlett, secretary-treasurer, Cameron.

Reeves-Ward-Winkler-Loving-Culberson-Hudspeth Counties Society

October 15, 1953

Common Eye Problems—Willis T. Carson, Odessa.
Tumors of Ear, Nose, and Throat—Jack L. Turner, Odessa.

Following a buffet dinner in Pecos on October 15 with members of the local woman's auxiliary, the Reeves-Ward-Winkler-Loving-Culberson Hudspeth Counties Medical Society held a business session and heard the program outlined above.

San Patricio-Aransas-Refugio Counties Society

December 1, 1953

(Reported by C. A. Selby, President)

Significance of Back Pain—Joseph Platz, Corpus Christi.

After a dinner in Sinton on December 1, the San Patricio-Aransas-Refugio Counties Medical Society heard the above scientific discussion and elected officers for 1954. New officers are Josephine Shelton, Refugio, president; Rose Tunnell, Taft, president-elect; Harry Miller, Refugio, vice-president; Maurice Horine, Ingleside, secretary-treasurer; John H. Finn, Refugio, delegate; and John Tunnell and Y. S. Jenkins, both of Taft, alternate delegates. L. G. Wood, Rockport; T. C. Meitzen, Refugio; and Dr. Jenkins were chosen as censors.

Dr. Selby, retiring president, and C. H. Simpson, both of Sinton, were hosts for the meeting. About twenty-five physicians attended.

Tarrant County Society

December 10, 1953

(Reported by Joseph F. McVeigh, Chairman, Public Relations Committee)

Approximately 200 persons attended the fourth in a series of open forums and panel discussions sponsored by the Tar-

rant County Medical Society on December 10 in the auditorium of the new Fort Worth Academy of Medicine. The subject was allergy, and Sim Hulsey was moderator. Panel speakers were William Flood, dermatologist; O. J. Emery, allergist; D. E. Neal, general practitioner; and Thomas J. Coleman, internist.

Of special interest to persons in Fort Worth was the question of fluoride in connection with allergy, since the city was scheduled to begin putting fluoride into the municipal water supply to prevent tooth decay among children. Dr. Coleman said that fluoridation is a controversial question, but so far as he had been able to determine, the evidence was in favor of fluoride. Fluoride in water is not known to be connected with any allergy, he stated. Dr. Hulsey pointed out that fluoride is similar to chlorine which has been used for years in water supplies.

The next forum was to be on poliomyelitis January 14. The first three forum subjects were menopause, accidents, and the common cold.

Washington-Burleson Counties Society

October 13, 1953

The Washington-Burleson Counties Medical Society met October 13 at the clinic of C. E. Southern in Brenham to hear the telephone postgraduate broadcast on obstetric emergencies. The broadcast was one in a series sponsored by the Texas Medical Association.

Webb-Zapata-Jim Hogg Counties Society

December 1, 1953

(Reported by A. S. McGee, Acting Secretary)

Your Medical Society—Mr. N. C. Forrester, Austin.

The Webb-Zapata-Jim Hogg Counties Medical Society met for dinner and an election of officers in Laredo on December 1. The woman's auxiliary met with the society.

The following officers were elected: V. L. Puig, Jr., president; M. E. Malakoff, vice-president; Paul H. Powell, Jr., secretary-treasurer; E. M. Longoria, delegate; and F. M. Canseco, censor. All new officers are from Laredo.

Mr. Forrester, executive secretary of the Texas Medical Association, discussed the Association's headquarters building and some of the problems that face the medical society today, including legislation, veterans' care, socialized medicine, segregation of white and Negro doctors, the relationship between the pharmacist and the medical doctor, and the nursing shortage. He was introduced by Dr. Malakoff, program chairman, and A. C. King, president, presided.

Wharton-Jackson-Matagorda-Fort Bend Counties Society

December 8, 1953

(Reported by Lorraine I. Stengl, Secretary)

On December 8 the Wharton-Jackson-Matagorda-Fort Bend Counties Medical Society met in El Campo for a Christmas buffet dinner with the local woman's auxiliary and then elected the following officers: C. V. Nichols, Richmond, president; E. B. Sanford, Palacios, vice-president; and Stanley E. Thompson, Richmond, secretary-treasurer. R. G. Johnson, New Gulf, was elected to the board of censors and Joseph Bader, Edna, to the adjudication board. Delegate is L. B. Johnson, El Campo; and Harold Northington, Wharton, and R. G. Johnson are alternates. R. G. Johnson is the legislative representative, and the two new members of the executive board are C. A. Shoultz, Bay City, and Franz Amman, Rosenberg.

DISTRICT SOCIETIES

Tenth District Society

October 3, 1953

Preemployment Examinations in Sabine Area Industries—William R. Thornton, Port Neches.

Industrial Burns and Their Treatment—Elliott B. Hay, Houston.

More Common Industrial Injuries of Bones and Joints—William H. Ainsworth, Galveston.

The semiannual session of the Tenth District Medical Society was held October 3 in Port Arthur, and the above scientific program was presented. Edmund D. Jones, Beaumont, president, presided.

AUXILIARY SECTION

ANNUAL AUXILIARY REPORTS

With little more than three months remaining before the Woman's Auxiliary to the Texas Medical Association holds its annual convention in San Antonio, Auxiliary officers and committee chairmen are being reminded that annual reports soon will be due.

County auxiliary presidents are requested to submit two main types of reports: (1) a statistical report and (2) a narrative report. The statistical report is a tabulation of all auxiliary activities and is made by filling out a blank form which will be sent in February, along with a letter of instruction, to county presidents by Mrs. Paul Brindley of Galveston, State Auxiliary Recording Secretary. "No single auxiliary," Mrs. Brindley points out, "will be expected to answer all questions. Only those pertaining to activities in which the group has participated need be answered." The completed statistical report should be returned to Mrs. Brindley by March 15.

The narrative report, also due March 15, is the basis of each county president's oral report to be given at the state convention. This report becomes a part of the convention transactions, printed yearly in the July issue of the JOURNAL. It is from the narrative reports, too, that the State Auxiliary President, Mrs. E. W. Coyle, obtains information for use in her report to the National Auxiliary.

County presidents are reminded to include mention of any outstanding activities or programs to be held after the report is submitted. "This is the place to let your star shine," says Mrs. Brindley. "Tell about your new projects, parties, or what-have-you, or your new and original way of doing the old jobs common to all auxiliaries. Make it good; make it interesting."

The following instructions for preparing narrative reports are given by Mrs. Brindley:

1. Type and double space the report on 8½ by 11 inch white paper and leave ample margins.

Officers of the Woman's Auxiliary to the Texas Medical Association: President, Mrs. E. W. Coyle, San Antonio; President-Elect, Mrs. Mark H. Latimer, Houston; First Vice-President (Organization), Mrs. John D. Gleckler, Denison; Second Vice-President (Physical Examinations), Mrs. Cecil O. Patterson, Dallas; Third Vice-President (Today's Health), Mrs. Ralph B. Payne, Amarillo; Fourth Vice-Presidents (Program), Mrs. Guy Knolle, Houston; Recording Secretary, Mrs. Paul Brindley, Galveston; Corresponding Secretary, Mrs. Charles L. McGehee, San Antonio; Treasurer, Mrs. Oscar M. Marchman, Jr., Dallas; Publicity Secretary, Mrs. Allen H. Neighbors, Jr., Austin; Parliamentarian, Mrs. H. S. Renshaw, Fort Worth.

2. Make an original and four clear carbon copies.

3. Send the original and one copy to Mrs. Paul Brindley, Recording Secretary, 4306 Sherman, Galveston; one copy to Mrs. E. W. Coyle, President, 213 Grant Avenue, Alamo Heights, San Antonio; and one copy to Mrs. Travis Smith, Historian, 849 Elmwood Drive, Abilene. Put one copy in the file for the incoming president.

At the 1954 convention, county presidents will be asked to limit their oral reports to two minutes each. Because of this time limit and the space limitations in the JOURNAL, length of narrative reports should be between 200 and 500 words, except in the case of auxiliaries having 100 or more members, which may use up to 1,000 words if necessary.

State officers and committee chairmen and council women also submit narrative reports at the annual convention. Rules and suggestions for preparing these reports are the same as those given for county reports except that the officer and committee reports are due April 1.

To obtain a breakdown on information of special concern to them, certain state officers and committee chairmen request county auxiliaries to send data on particular projects direct to them for use in their reports. The following deadline for these additional reports have been given:

February 1—Mental health survey data. Send to Mrs. A. B. Pumphrey, 100 Pumphrey Drive, Fort Worth.

February 1—Library Fund donations. Send to Mrs. S. F. Harrington, 3722 Cragmont, Dallas.

February 15—Report on number of physical examinations for physicians and families of physicians. Send to Mrs. Cecil O. Patterson, 3537 Southwestern Boulevard, Dallas.

March 1—Report on most interesting meeting of the year for Archives. Send to Mrs. F. F. Kirby, 2801 Sanger Avenue, Waco.

March 10—History of county auxiliary. Send to Mrs. Travis Smith, 849 Elmwood Drive, Abilene.

March 10—First three prize winning essays in AAPS essay contest from each county auxiliary. Send to Texas Medical Association, 1801 Lamar Boulevard, Austin, for judging for state prizes.

April 1—Report on number of *Bulletin* subscriptions sold since May 1, 1953. Send to Mrs. E. H. Marek, Yoakum.

April 30—Report on number of *Today's Health* subscriptions sold. Send to Mrs. Ralph B. Payne, 87 North Avondale, Amarillo.

Mrs. O. M. Marchman, Jr., State Auxiliary Treasurer, has placed in the mail to each county treasurer a letter plus a four-page instruction pamphlet from the National Auxiliary. Dues for former members are being accepted by Mrs. Marchman, 5539 Montrose, Dallas, from January 15 to March 1. New members' dues may be accepted until April 1. Donations to the philanthropic funds (Library, Student Loan, American Medical Education Foundation, and Memorial) go directly to the respective chairmen and are counted in the current year up until the treasurer's books are closed—about April 15. Vouchers from state officers and committee chairmen must be submitted to Mrs. Marchman by March 31.

COUNTY AUXILIARIES

"Medicine and Magic in Childbirth" was reviewed by Mrs. W. Frank Armstrong, Fort Worth, for the November 6 meeting of *Navarro County Auxiliary*. Turning to the latter end of life for its business session, the group voted to remember the aged at "Twilight Home" with Christmas gifts instead of exchanging gifts among its own membership, and a committee was appointed to formulate plans for a more permanent program of assistance to residents of the home. A dessert course was served by Mesdames Leslie Kelton and Bernard Rosen, Corsicana.

Recruitment of high school girls to enter nurses' training was discussed by *Gray-Wheeler-Hansford-Hemphill-Lipscomb-Roberts-Ochiltree-Hutchinson-Carson Counties Auxiliary* in the home of Mrs. W. W. Brooks, Borger, October 14. Mrs. Claunch Brindley was named chairman of the recruitment committee and Mrs. Larry Hanson, co-chairman; both reside in Borger.

Mrs. E. W. Coyle, San Antonio, President of the Woman's Auxiliary to the Texas Medical Association, was guest speaker at a dinner given by the *Smith County Auxiliary* to honor doctors of the area October 17. Dr. Coyle also was present for the Tyler event. Mrs. Coyle likewise addressed the *Gregg County Auxiliary* at a luncheon meeting October 15 in Kilgore.

Hunt County Auxiliary entertained its medical society at dinner October 13 in the home of Dr. and Mrs. J. W. Ward, Greenville. An original skit under the direction of Mrs. E. T. Crim and piano selections by the daughter of Dr. and Mrs. Grady Bruce entertained guests.

A Christmas party was held by *El Paso County Auxiliary* in El Paso December 14 in connection with a luncheon for which Mesdames Frank O. Barrett and Harry H. Varner were chairmen. Mrs. Haskell D. Hatfield read two original poems, and a cake walk was conducted by Mrs. Russell Deter and Mrs. Joe Carter, cakes, cookies, and candies made and donated by members being prizes for the event. The auxiliary raised funds to help finance its nurse scholarship, nurse recruitment, hospital work, and other projects.

Tarrant County Auxiliary members and guests learned the art of candlemaking and Christmas decoration at a guest day luncheon December 11 in Fort Worth.

The purposes and function of standing committees were reviewed by the *Harrison County Auxiliary* in Marshall early in October. Members heard from the following chairmen: Mesdames Maurice Murphy, Roger Harmon, Malcolm McNatt, Ray H. Carter, and John E. Hill. Mrs. George Bennett was in charge of the program.

Mrs. D. C. Bennett, Orange, president of the *Orange County Auxiliary*, outlined her plans for the year when the group met for luncheon October 13 in Orange. The main project is to be work for a treatment center for children with cerebral palsy.

October brought social events for the *Galveston, Dallas, Nueces, and Travis Counties Auxiliaries*. A luncheon in Galveston on October 18 honored Mrs. E. W. Coyle, San Antonio, President of the State Auxiliary; Mrs. W. D. Nicholson, Freeport, District 8 council woman; and Dr. Jesse B. Johnson, Galveston, president of the local county medical society. Officers of the Dallas County Auxiliary were honored at a coffee October 7 in Dallas. Honorees included Mrs. Sidney Baird, president; Mrs. Speight Jenkins, immediate past president; Mrs. Robert Sparkman, president-elect; and members of the board. Twelve new members of Nueces County Auxiliary were special guests at a coffee in the home of Mrs. Charles Mella, Corpus Christi. Mrs. Charles Clark was chairman of arrangements with Mrs. Thomas Edwards as co-chairman. The Travis County Auxiliary honored sixteen new members at a coffee October 20 in Austin, at which time W. A. Stiegler, a publisher, reviewed a book of which an auxiliary member, Mrs. Walter Goddard, is co-author.

POLIO MOVIES AVAILABLE

Auxiliary members are urged by Mrs. E. W. Coyle, San Antonio, State Auxiliary President, to take an active part in the 1954 Mothers' March on Polio campaigns currently under way.

Several films have been made available by the National Foundation for Infantile Paralysis for loan to Auxiliary members. They include "Marbles and Lollipops," an account of gamma globulin field tests; "1953 Newsreel," showing

how polio epidemics are fought; and "1954 Mothers' March on Polio," featuring Miss Helen Hayes. All of the films are 16 mm. sound reels running ten minutes each. Information on how to obtain the films may be had by writing to Mrs. Arleen Draker at the Association's central offices in Austin.

Mrs. Coyle is a member of the March of Dimes State Advisory Committee and represented the Auxiliary at the committee's December 12 meeting in Austin.

AUXILIARY DEATHS

Mrs. W. H. Brandau, Beaumont, a former president of the Woman's Auxiliary to the Jefferson County Medical Society, died November 8, 1953, several days after suffering a cerebral hemorrhage. An active civic and social worker, Mrs. Brandau is survived by her husband, her mother, a daughter, a brother, and a granddaughter.

DEATHS

E. H. CARY

Dr. Edward Henry Cary, Dallas, Texas, past president of the Dallas County Medical Society, Texas Medical Association, Southern Medical Association, and American Medical Association and former dean of Baylor University College of Medicine, died December 11, 1953.

A native of Union Springs, Ala., Dr. Cary was born February 28, 1872, the son of Joseph Milton and Lucy Janette (Powell) Cary. He came to Texas as a young man to work with his brother. Later he obtained his medical education in New York, being graduated from Bellevue Hospital Medical College in 1898, interning at Bellevue Hospital, and then serving at the New York Eve and Ear Infirmary, during which time he was chief of the Eye Clinic of Bellevue Dispensary and substitute professor of ophthalmology in New York Polyclinic. Returning to Texas in 1901, Dr. Cary became professor of ophthalmology and otolaryngology and head of the department first at Dallas Medical College and then, when the old school became part of Baylor University, at Baylor University College of Medicine. He also had been dean of Dallas Medical College and was instrumental in arranging for its absorption by Baylor. He immediately was named dean of the new college and served in that capacity from his appointment in 1903 until 1922; from 1929 until 1943 he was dean emeritus. He was chairman of the Department of Surgery from 1921 to 1929, chairman of the faculty from 1909 to 1929, and chairman of the advisory board of Baylor School and Hospital from 1909 to 1929. Under his direction was organized the Baylor Medical and Surgical Unit which served in France during World War I. He was chairman of the district medical advisory board at that time and was a member of the district appeal board of Selective Service during World War II. In 1939 Dr. Cary helped to organize and became president of the Southwestern Medical Foundation, which was designed to support a great medical center in Dallas. Failure to make agreeable arrangements with Baylor University College of Medicine, which soon moved to Houston, did not stop Dr. Cary and his associates, who assisted in establishing Southwestern Medical College in 1943 and have continued support of the latter institution since its inclusion in the University of Texas system.

When Dr. Cary first returned to Texas after completing his medical studies, he took over the management of A. P. Cary surgical supply company which had been founded by his recently deceased brother, and he continued as president of that business. He likewise was president of the Cary-Schneider Investment Company and of the Medical Arts Hospital of Dallas. In 1921 he built the Medical Arts Building, the first skyscraper especially for offices of physicians. He also was president of Group Hospital Service, the Texas affiliate of Blue Cross, which he had helped to launch as a hospital insurance plan for Dallas school teachers. He was president also of the Kessler Plan Association and a director of the Republic National Bank of Dallas.

Dr. Cary found time to practice his specialty of eye, ear, nose, and throat despite his many educational, business, and civic interests and wrote numerous articles in that field. In addition to holding membership in county, state, regional, and national medical associations, he was the first president of the Texas Society of Ophthalmology and Otolaryngology, a member of the American Laryngological, Rhinological, and Otological Society and the American Academy of Ophthalmology and Otolaryngology, and a fellow of the American College of Surgeons. President of the Texas Medical Association for the 1917-1918 term, Dr. Cary was elected to emeritus membership in 1948. He had represented Texas in the

DR. E. H. CARY

House of Delegates of the American Medical Association off and on over a period of forty years and was a trustee of the AMA from 1925 to 1929 before becoming president for 1932-1933, the only AMA president Texas has had. One of the first to recognize the dangers of socialized medicine, Dr. Cary was president of the National Physicians Committee from its inception until the need for its fight against socializing forces brought its liquidation.

Dr. Cary was president of the Dallas Historical Society and of the Texas Philosophical Society, and a member of the Masonic Order (a thirty-second degree Mason and member of the Shrine), Phi Alpha Sigma, Alpha Omega Alpha, Society of Colonial Wars, First Families of Virginia, Critics Club, Dallas Athletic Club, Brookhollow Club, and Dallas Country Club.

An obituary ordinarily will not be published more than four months after date of death. Cooperation in reporting deaths of physicians and in furnishing appropriate biographical material promptly is solicited.

In addition to honoring him with positions of leadership in numerous organizations, Dr. Cary's associates paid more specific tribute to him on several occasions. He was awarded the honorary degree of doctor of laws by Baylor University in 1916. A bronze bust of him was presented to Baylor University in 1929. He was honor guest at a formal dinner given by Dallas County Medical Society, the Woman's Auxiliary to Dallas County Medical Society, the Dallas County Dental Society, and the Kessler Plan Association in 1931 upon his election as President-Elect of the AMA. He was presented the Linz award as Dallas' most outstanding citizen in 1945. He was given another dinner to celebrate his seventy-fifth birthday in 1947, at which time a $20,000 gift was made in his name for scholarships at Southwestern Medical School.

In 1948 a biography of Dr. Cary, "More Than Armies," was written by Booth Mooney. In the introduction, Dr. Morris Fishbein said of Dr. Cary: "This man is a physician, a distinguished scientist, a medical statesman, a great builder, educator, idealist—all combined in the person of one dynamic, driving, courageous and at the same time gentle, friendly and affectionate human being."

Dr. Cary is survived by his wife, the former Miss Georgie Fonda Schneider, whom he married April 19, 1911, in Dallas, the first president of the Woman's Auxiliary to the Texas Medical Association and of the Woman's Auxiliary to the Southern Medical Association. Also surviving are a son, E. H. Cary, Jr., Dallas; three daughters, Mrs. Ogden Brower, North Tarrytown, New York; Mrs. W. L. Carwile, Jr., Oklahoma City; and Mrs. John I. Hill, Houston; a sister, Mrs. Lucile Cary Lowry, Dallas; and a brother, Joseph M. Cary, Houston.

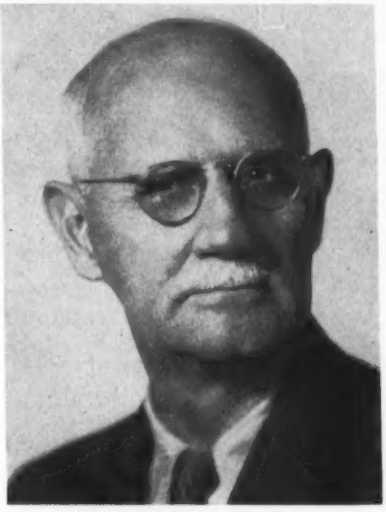
M. L. GRAVES

Dr. Marvin Lee Graves, a former president of the Texas Medical Association, died November 19, 1953, in Houston, Texas.

The son of the Rev. George W. and Mary Priscilla (Fowler) Graves, Dr. Graves was born in Bosqueville, March 26, 1867. Two years before beginning his premedical studies under Dr. H. C. Ghent of Belton, he served as principal of Belton High School. He received a bachelor's degree in 1885 and a master's degree in 1891 from Bellevue Hospital, New York, now a part of New York University. Dr. Graves held honorary degrees from Southern Methodist University, Dallas, and Baylor University College of Medicine, Houston, as well as an honorary doctor of science degree awarded him in 1949 on the 190th anniversary of Southwestern University, Georgetown.

After receiving his medical degree, Dr. Graves moved to Waco where he practiced for seven years before moving to San Antonio to serve as superintendent of Southwestern Hospital for the Insane. In Waco he was physician for the Methodist Orphanage. In 1950, he opened a practice in Galveston and became professor of medicine and nervous and mental diseases at the University of Texas School of Medicine and physician-in-chief at John Sealy Hospital. More recently he has been emeritus professor of medicine at the Medical Branch in Galveston. He practiced in Galveston for twenty years before resigning his professorship and moving to Houston in 1925 to continue private practice. In Houston he was a member of the Methodist Hospital's executive board and later became an honorary member of the hospital staff. Following his retirement in 1949, Dr. Graves became medical adviser to the American General Insurance Company.

Dr. Graves was a member of the American Medical Association and Texas Medical Association through the Harris County Medical Society. A member of the State Association for forty-eight years, he was elected to emeritus membership in 1937. He served as President of the Texas Medical Association for 1913-1914, was a member of the Board of Trustees for ten years, and served as chairman of several scientific sections for Association annual sessions. He had been president of the Houston Society of Internal Medicine, Texas Club of Internists, Texas Association of Life Insurance Medical Directors, and the Texas Heart Association. He was a fellow of the American College of Physicians and a member of the Southern Medical Association. He was a member also



DR. MARVIN L. GRAVES

of the Phi Chi medical fraternity. He was a member for twenty-eight years of the Board of Stewards of the First Methodist Church of Houston, and for twenty-one years before that, held the same office in the First Methodist Church of Galveston. He attended the Methodist Ecumenical Conference in London in 1921. He was a member of the Masonic Lodge, Chamber of Commerce, and Houston Country Club.

In 1893, Dr. Graves married the former Miss Laura Ghent of Belton. In addition to his wife, who served as a president of the Woman's Auxiliary to the Texas Medical Association, he is survived by the following: a son, Dr. Ghent Graves, Houston; two daughters, Mrs. Laura Lee Steel and Mrs. Mary Alice Morse, both of Houston; two sisters, Miss Minnie B. Graves and Mrs. Gladys Stevenson, both of Waco; and five brothers, Newton Graves, Augusta, Ga.; E. F. Graves, Waco; and Judge George Graves, Ralf Graves, D.D.S., and P. F. Graves, all of Houston.

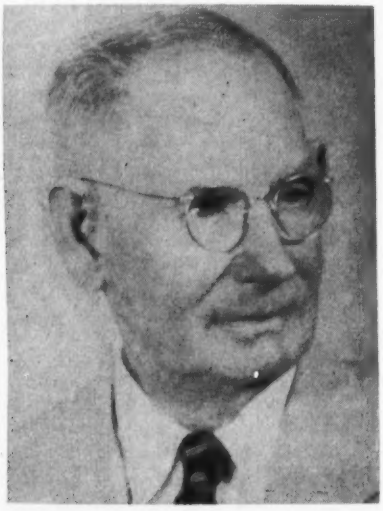
J. S. BUIE

Dr. James Shaw Buie, Mertens, Texas, died November 11, 1953, in a Waco hospital of coronary heart disease.

Born March 1, 1879, near Brandon, Hill County, Texas, the son of Dr. Whitefield and Julia (Shaw) Buie, Dr. Buie attended the old Southwestern University Medical College in Dallas, where he received his medical degree in 1904. He then interned at St. Paul's Hospital in Dallas. Dr. Buie practiced in Hill County for thirty years, moving from Brandon to Hillsboro and later to Mertens. For thirteen years he was connected with the Texas State Hospital system, working at Terrell, Rusk, Austin, and Mexia. While in Mexia he was

with the Mexia State School and Home. Dr. Buie became a member of the staff of the Central Oklahoma State Hospital, Norman, Okla., in 1951. In 1953, he retired from this position and returned to his home in Mertens, where he lived until his death.

Dr. Buie was a member of the American Medical Association and Texas Medical Association through Hill County Medical Society, and also of the Texas Academy of General Practice. He was a member of the Mertens Masonic Lodge and the Presbyterian Church.



DR. J. S. BUIE

Dr. Buie married Miss Ethel Cooper in 1906. His first wife preceded him in death in 1941. In 1945, he married the former Mrs. Georgia Pearl Elliott. She and the following survive him: a daughter, Miss Mary Frances Buie of Terrell; two sisters, Mrs. Julia Mae Wilhelm of San Antonio and Mrs. Margaret P. Williams of Terrell; two nephews, Willie S. Williams of South Houston and Judge Sterling Williams of Snyder.

H. D. KEARBY

Dr. Harold Donald Kearby died November 21, 1953, at Houston, Texas, of coronary infarction.

Dr. Kearby was born September 27, 1909, at Helena, Mo., the son of Dr. Howard Denton and Verda Eva Kearby. He attended St. Joseph Junior College, St. Joseph, Mo.; the University of Missouri, Columbia; and the University of Arkansas, Fayetteville, and received his medical degree from the University of Arkansas School of Medicine, Little Rock, in 1938. After interning at St. Margaret's Hospital, Kansas City, Kan., and serving a residency in urology at the Turner Urological Institute, Houston, Dr. Kearby specialized in urology at Houston until he entered the United States Air Force in 1951. A major, he had served as chief of urology at Lackland Air Force Base, San Antonio, and as head of the department of urology at the largest Air Force hospital in the Far East. He was recently discharged from the Air Force and had returned to Houston.

Dr. Kearby was a member of the American Medical Association and Texas Medical Association through Harris County Medical Society. He also held membership in the Postgraduate Medical Assembly of South Texas, South Central Section of the American Urological Association, Texas Urological Society, Phi Beta Pi medical fraternity, and Woodmen of the World, and was a member of the Baptist Church.

Dr. Kearby is survived by his wife, the former Miss Geraldine Arnold, whom he married July 30, 1934, in Lawrence, Kans., and his grandmother, Mrs. Belle Holland of St. Joseph, Mo.

J. H. BROWN

Dr. Joseph Hyal Brown died October 19, 1953, in Fort Worth, Texas, after suffering a heart attack.

Dr. Brown was born in Hillsboro, July 9, 1901. He received his academic education at Texas Agricultural and Mechanical College, College Station; Texas Christian University, Fort Worth; and Tulane University of Louisiana, New Orleans. He was graduated in medicine from Tulane University in 1930, and served his internship at St. Joseph's Infirmary in Fort Worth.

Dr. Brown was division surgeon for Rock Island Lines and Southern Pacific Lines and was in general practice, being associated for some years with his father-in-law, Dr. Will C. Duringer. He was a current member of the American Medical Association through Tarrant County Medical Society.

Survivors include his wife, the former Miss Dorothy Duringer; a 2½ year old daughter, Dorothy Duringer Brown; and his mother, Mrs. Maud A. Brown, all of Fort Worth.

J. A. RIEBEL

Dr. John Alan Riebel, Kingsville, Texas, died September 12, 1953, in that city.

Born in Columbus, Ohio, August 29, 1907, Dr. Riebel, whose father, brother, and uncle also were physicians, received his education at Ohio State University and the University's College of Medicine, Columbus, from which he was graduated with a medical degree in 1934. He interned at the White Cross Hospital in Columbus, after which he entered private practice in Columbus and later with his uncle practiced in Ashland, Ohio, where he remained until he joined the Medical Corps of the United States Army Air Force in 1941. During his five years in the service, Dr. Riebel was a flight surgeon in England and Africa and was stationed in Texas twice. He returned to private practice in Ashland until 1949, when he became chief surgeon at the United States Veterans Administration Hospital in Lexington, Ky. He moved to Kingsville in 1952. He was a general practitioner and a surgeon.

A member of the American Medical Association and the Texas Medical Association through the Kleberg-Kenedy Counties Medical Society, Dr. Riebel formerly had been a member of the Ohio State Medical Association. He belonged to the Presbyterian Church.

Dr. Riebel's wife and two daughters, Dawn Riebel and Dusk Riebel, survive.

F. L. ROBBINS

Dr. Frank Leonard Robbins, Baytown, Texas, died November 18, 1953.

Born in Freelandville, Ind., May 29, 1881, the son of Mr. and Mrs. Thomas Robbins, Dr. Robbins received his preliminary education in Vincennes University, Vincennes, Ind. He attended Louisville Medical College, where he was graduated in 1906. Dr. Robbins externed at Good Samaritan Hospital, Lexington, Ky., in 1905. He practiced in Carlisle, Ind., for eleven years, Goose Creek, Texas, for thirty-seven years, and Baytown from 1944 until his death. He was city health officer at Goose Creek and head of the Welfare Clinic of Baytown for several years.

Dr. Robbins was a member of the American Medical Association and Texas Medical Association through Harris County

Medical Society, and of the Houston Academy of Medicine. In 1952, he was elected to honorary membership in the Texas Medical Association. While practicing in Indiana, he belonged to the Sullivan County Medical Society. Dr. Robbins was a captain in the Medical Corps of the American Expeditionary Force during World War I. He was a charter member of the Baytown Rotary Club, a Mason, and a member of the Episcopal Church.

In 1909 Dr. Robbins married the former Miss Lena M. Grizzle in Carlisle, Ind. His wife survives as do two sons, Philip L. Robbins and Ruel H. Robbins, both of New Iberia, La., and a daughter, Miss Helen M. Robbins, Baytown.

F. S. REASONOVER

Dr. Farley Straughn Reasonover, Grand Prairie, Texas, died at the home of his parents in Dallas on October 25, 1953, as the result of an accident.

He was born to Mr. and Mrs. Farley Reasonover in Denison on February 27, 1926. Dr. Reasonover acquired his preliminary education at Highland Park High School, from which he was graduated with honors; Oklahoma Agricultural and Mechanical College, Stillwater; the University of California at Los Angeles; and the University of California, Berkeley. After his graduation from Southwestern Medical

DR. FARLEY S. REASONOVER

School of the University of Texas in June, 1952, Dr. Reasonover served an internship in Baylor Hospital. He had begun the practice of medicine in Grand Prairie this past July.

During World War II Dr. Reasonover received Army basic training at Camp Wolters, Texas, and was affiliated with the Eighty-Fifth Mountain Infantry Regiment and the Tenth Mountain Ski Troops. He was a medical clerk. During nine months' overseas duty he took part in the Italian campaign and was awarded a bronze star citation.

A member of the American and Texas Medical Associations through Dallas County Medical Society, Dr. Reasonover was a member of Phi Chi medical fraternity, the National Honor Society, and Alpha Tau Omega fraternity. He was affiliated with the Methodist Church.

Survivors are his parents, Mr. and Mrs. Farley Reasonover, Dallas.

G. L. CARLISLE

Dr. George Lawson Carlisle, Dallas, Texas, died of coronary occlusion in his home November 2, 1953.

Dr. Carlisle was born September 7, 1891, in Jackson, Miss., to George W. and Virginia (Fearn) Carlisle. He attended Dallas High School and Millsaps College in Jackson and was graduated in 1914 from Southern Methodist University Medical Department, Dallas. After internships at St. Paul's Hospital, Dallas, and St. Mary's Infirmary, Galveston, Dr. Carlisle was a resident in cardiology at Massachusetts General Hospital, Boston. Since 1915 he had practiced in Dallas with the exception of two years' military service as first lieutenant in the United States Army during World War I. Certified by the American Board of Internal Medicine, Dr. Carlisle was particularly interested in heart diseases.

The problem of lay health education was one to which Dr. Carlisle devoted a great amount of effort. As George Lawson, M. D., he was co-author with his wife, Helen Parker, R. N., of a health column "Your Health" which appeared thrice weekly in the woman's section of the Dallas *News*. For a time he also conducted a program on medical topics over a Dallas radio station, KRLD, and he wrote a book for general practitioners entitled "Practical Talks on Heart Diseases." Dr. Carlisle earned a reputation as a philosopher as well as a scientist. His interest in the patient as a person and his understanding of sick people's problems, which were evident in his writings, earned for him a patient's comment that he was a "modern adaptation of the old-fashioned family physician."

DR. GEORGE LAWSON CARLISLE

A member for thirty-eight years of the Texas and American Medical Associations, Dr. Carlisle in 1927 served as president of the Dallas County Medical Society. He was a member as well of the Dallas Southern Clinical Society, Dallas Academy of Internal Medicine, and Texas and American Heart Associations. Dr. Carlisle was professor of physical diagnosis at Baylor University College of Medicine while it was located in Dallas and taught at Southwestern Medical School of the University of Texas, being a consultant in medicine there at the time of his death. He had contributed numerous scientific papers to national medical journals. In

1945 he was president of Baylor University Hospital staff. He was a member of Theta Kappa Psi medical fraternity and Pi Kappa Alpha fraternity and was affiliated with the Methodist Church.

Dr. Carlisle married Miss Mildred King in Dallas in 1915; their children, Mrs. Donald Gay, Dallas, and George L. Carlisle, Jr., Austin, and two grandchildren survive. Also surviving are a second wife, the former Miss Helen Parker, whom he wed January 16, 1949, in Dallas; and three brothers, Hugh P. Carlisle, G. Fern Carlisle, and John B. Carlisle, all of Dallas.

C. C. CARRICO

Dr. Carl C. Carrico, Houston, Texas, died in a Houston hospital September 7, 1953, of cerebral thrombosis.

Born August 30, 1901, in Dallas, the son of the late B. J. and Effie Mae Carrico, Dr. Carrico attended the University of Texas, Austin, and was graduated in 1928 from Baylor University College of Medicine, then in Dallas. After interning at Gorgas Hospital, Panama Canal Zone, he practiced at Scott and White Clinic, Temple, and served for a year at Huntsville as a hospital physician for the Texas Prison System. He then moved to Houston, where he was the physician for the Post Office for eleven years and a general practitioner until his death, with the exception of

DR. CARL C. CARRICO

the period between 1941 and 1946, during which time he was a captain in the United States Army Medical Corps and was chief of the communicable disease section of the Harmon General Hospital at Longview. With the Forty-Fifth Infantry Division, Dr. Carrico participated in the invasions of Sicily and Salerno and the Anzio beachhead. He was a favorite of the late Ernie Pyle, who referred to Dr. Carrico in news dispatches and later in his book, "Brave Men," as "the red-headed doctor." Dr. Carrico was on the staffs of St. Joseph's Infirmary and Methodist and Hermann Hospitals in Houston.

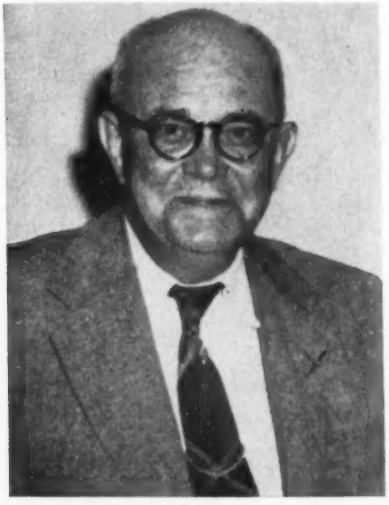
Elected to honorary membership in the Texas Medical Association through the Harris County Medical Society in 1950, Dr. Carrico was a member of the American Medical Association, Southern Medical Association, Postgraduate Medical Assembly of South Texas, Texas Academy of General Practice, and the American Academy of General Practice. He also belonged to the American Legion, Elks, and National Association of Retired Civil Service Employees.

Dr. Carrico married Miss Helen M. Murphy on August 4, 1934, in Denison. She survives him as do a son, Carl C. Carrico II, and a daughter, Miss Carolynn Murphy Carrico, both of Houston, and a sister, Mrs. J. K. Wilson, Los Angeles.

E. W. LOOMIS

Dr. Edgar Webb Loomis died November 18, 1953, at a Dallas, Texas, hospital.

Born in Dallas, February 21, 1886, he was graduated from the University of the South, Sewanee, Tenn., and received his medical degree from Southwestern University Medical College, Dallas, in 1910. After graduation, he served in Alaska as a medical missionary for the Episcopal Church. Upon his return to Dallas in 1912, he became assistant city health officer and later director of the Health Department. He resigned his post to accept a commission in the Medical Corps of the United States Army during World War I and served two and one-half years in France as a major. He returned to Dallas and his health department office after the war but resigned again in 1922 to begin private general practice. Several years later Dr. Loomis took special work at the Babies Hospital and Bellevue Hospital in New York and limited his practice to diseases of infants and children when he returned to Texas. One of the first Dallas doctors to specialize in pediatrics, he was admitted to the American Academy of Pediatrics in 1934 and was certified by the American Board of Pediatrics.



DR. EDGAR W. LOOMIS

Dr. Loomis was a member of the American Medical Association and Texas Medical Association through Dallas County Medical Society. He belonged to the Episcopal Church and Hella Temple Shrine.

His wife, the former Miss Addie Jane Walker of Quanah, whom he married on March 15, 1922, died February 21, 1936.

Surviving Dr. Loomis are three daughters, Mrs. J. P. Butler and Mrs. R. E. Collier, both of Dallas, and Mrs. C. R. Bryant of College Station; two brothers, Richard F. Loomis and John S. Loomis, both of Dallas; and four granddaughters.